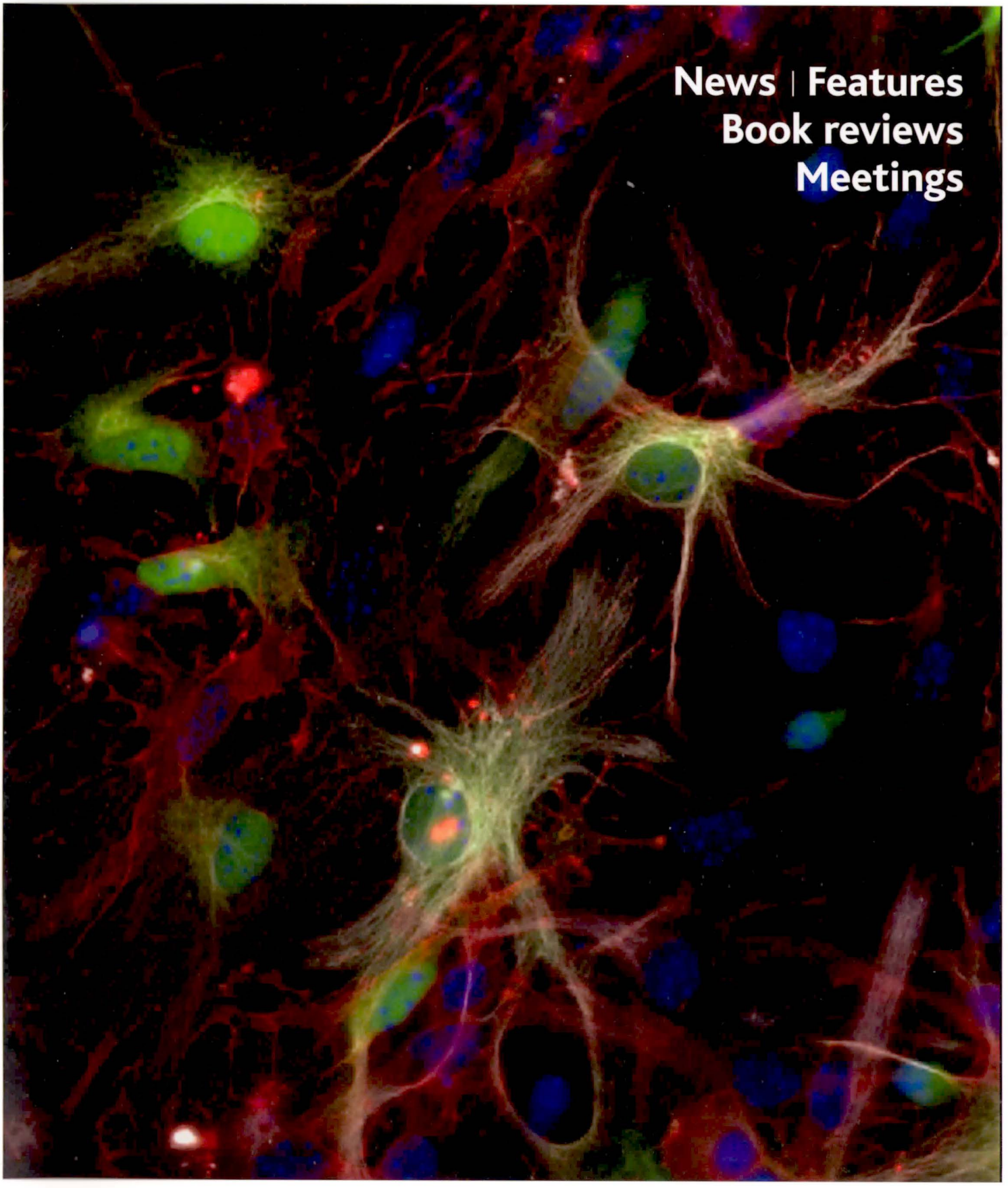
SPRING 2010

BSCB Newsletter

BRITISH SOCIETY FOR CELL BIOLOGY



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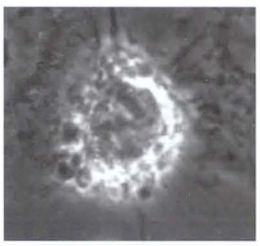
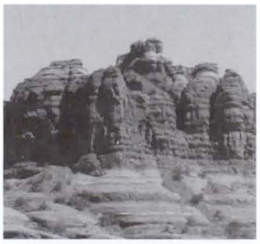


Editorial

Welcome to the Spring 2010 issue of the BSCB newsletter. Publication coincides with the 2010 Spring meeting in Warwick – a joint meeting with BSDB with a fantastic speaker list with a strong focus on disease mechanisms and stem cell technology. The Autumn meeting this year will take place in Oxford and is focussed on the organization of cells through the cell cycle. Again, a great list of speakers promises much for this exciting meeting.

This issue includes the usual range of meeting reports as well as reports from our summer studentship awardees from last year. This scheme will be running again in 2010 – full details will be on the website. Similarly we profile the winner of our Science Writing Competition who will be awarded her prize at the Spring meeting. We are also pleased to announce the inaugural BSCB Image Competition for which we hope to get your very best images reflecting cell biology research in all its forms. In addition to an excellent article from Martin Ziedler on life after a PhD, it is with sadness that we include a piece reflecting on the life and career of Emmanuelle Caron who sadly died last year. Many members will have known Emmanuelle and we hope that this piece from Vania Braga and Anne Ridley provides a fitting tribute.

Our regular PhD student contributor Jay Stone has written about the important subject of the libel laws and how they relate to science. The BSCB has recently signed up in support of the Keep Reform Campaign making this a timely and important piece.

This issue also marks my last as Editor. Kate Nobes (Catherine.Nobes@bristol.ac.uk) will be taking over this role with immediate effect and so all future correspondence, suggestions for content, comments and queries should be addressed to her. I have thoroughly enjoyed my role as editor and would like to thank all those contributors who have made this a pleasurable job. In particular I would like to thank Giles Newton at the Wellcome Trust who does the layout for each issue and transforms each collection of content into the pages you see for each issue. The newsletter is a forum for the membership so please do email Kate with any suggestions for future articles.

The Editor: David Stephens
University of Bristol
david.stephens@bristol.ac.uk

The cover image shows a population of eGFP astrocytes prior to transplantation. Astrocytes were labelled with GFP (green), GFAP (red) and GLAST (white) with nuclei identified using Hoescht (blue). Images was acquired using Leica DM6000 light microscope with 40x Leica lens. The image was taken by Dan Webber (Cambridge) and you can read his report on the 39th Annual meeting for the Society for Neuroscience in the meeting reports section.

Newsletter editor: David Stephens **Production:** Giles Newton **Website:** www.bscb.org **Printer:** Hobbs

News

BSCB Newsletter Cover Image Competition

The BSCB is pleased to announce the inauguration of an image competition. Entries should illustrate cell biology in any form and the winning images will be used as cover art for the newsletter.

The closing date for entries for the 2010 competition is: 30th June 2010. Please see the full rules and entry requirements below. You must be a current member of BSCB to enter.

Eligibility

1. This competition is open to members of the British Society for Cell Biology. Entrants must be a member at the time of submission of entries.

2. Only one entry per person is allowed.

3. The subject matter of competition entries is flexible but must reflect current research in Cell Biology.

Submission

1. Entrants must supply their name, address, email address, and BSCB membership number on entry.

2. Entries must be sent by email (10 x 11.96 cm 300 dpi) to Paul Andrews (bscbimagecomp@googlemail.com). Shortlisted entries will be requested on CD as 600 dpi JPG saved at maximum resolution sized at 196 mm wide x 230.5mm high and in RGB colour mode. At the time of submission, entrants must state clearly that they are the creator of the submitted image.

3. Your entry should adopt the file name initial_surname.jpeg e.g. a_einstein.jpeg.

4. Entrants should supply a concise stand-alone caption limited to 50 words as a MS Word document on the same CD, labelled initial_surname_caption.doc.

5. The deadline for entries is June 30th 2010)

6. Entries that do not conform to the entry requirements will be disqualified.

Prizes

Prizes will be awarded as follows: 1st Prize £100, 2nd Prize £75, 3rd Prize £25

General information

1. Entries will be anonymized prior to judging.

2. The organisers reserve the right to cancel this competition at any stage, if deemed necessary in their opinion, and if circumstances arise outside their control.

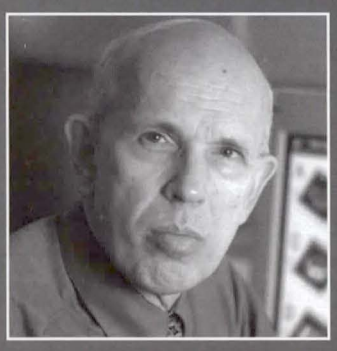
3. The organisers' decisions are final in every situation and no correspondence will be entered into.

4. Entries will be published on BSCB webpages and will also be used to illustrate BSCB newsletters and other promotional material. Copyright will remain with the creator. If you do not agree that images may be used as stated you must stipulate this on the entry form.

5. Entrants will be deemed to have understood the competition rules and accepted them and agree to be bound to them when entering the competition.

New Director for the Cardiff School of Biosciences

The distinguished physiologist Professor Ole Petersen CBE, FRS is the new Director of the School of Biosciences, Cardiff University. During his research career, Professor Petersen has made major breakthroughs in understanding the regulatory role of calcium in cell physiology and its clinical implications, including in pancreatic cancer and the relationship of alcohol and pancreatitis.



Prof Petersen will continue his exploration of these disease mechanisms, and has transferred his MRC Professorship, which he previously held at Liverpool University.

He has more than 300 academic articles to his name and more than 16,000 citations. He served as Vice-President of the Royal Society in 2005–6 and has just completed a 9-year term as Secretary-General of the International Union of Physiological Sciences.

He is currently Chair of the European Research Council's starting grant panel for physiology, pathophysiology and endocrinology and is keen to develop awareness of European funding, believing "it's a very good thing for there to be a European-wide body where competitive grants are made purely on the basis of research excellence."

2010 Members Subscriptions

This is a general notice to say that subscriptions for 2010 collected by Direct Debit will be collected either on 20th April (for membership numbers up to 2755) or 5th October (for membership numbers after 2755). An e-mail reminder will be sent just prior to these dates. Please inform the Treasurer Adrian Harwood (harwoodaj@cf.ac.uk) of any changes your subscription details prior to these dates.

Thank you for supporting the BSCB.

Libel reform campaign

The BSCB has signed the libel reform campaign petition following a request by Sense about Science's Keep Libel Laws out of Science.

Sense about Science has joined up with the free speech organisations Index on Censorship and English PEN to form a coalition for libel reform, which is calling for a wider public interest defence to protect writers and scientists tackling important subjects that are in the public interest as well as ensuring public access to these articles. Please see www.senseaboutscience.org.uk/index.php/site/project/333/ for more information.

By coincidence, our regular contributor, Jay Stone, also chose to write about this topic in this issue. You can reader her piece in the PhD student section of this newsletter. Here, Jay provides some background to the whole issue and highlights why it is a subject with which we should engage.

In order to be taken seriously by politicians a petition such as this needs to have approximately 100,000 signatures. If you would like to sign as an individual, please visit www.libelreform.org.

2009 UK International Fellowship Association E:Survey

The final report of the RCUK 2009 UK International Fellowship Association E:Survey have been published today on the RCUK website. The survey, run by RCUK, was developed to find out whether a UK International Fellowship Association should be established. Fellows were asked how they currently keep in touch with colleagues and research associates and what existing academic networks they use. The survey was also interested in how Fellows find out about funding opportunities, with a particular focus on international collaborations, and how they access research career advice, such as guidance helpful when moving to the UK or overseas for an academic position.

RCUK and its partners are now using the survey findings to look into how best to serve the needs of Fellows both in the UK and internationally.

With over 93% of respondents in regular contact with former colleagues and research associates, Fellows are clearly making the most of the wide range of communication methods available to stay in touch. One key area highlighted by the survey was the need for a single source announcing funding opportunities in the UK. By developing a website, or portal, listing research career opportunities in the UK, respondents said they would be less likely to miss out on funding and it would reduce time spent and simplify the process of searching for opportunities.

A copy of the survey report is available to download on the RCUK website at www.rcuk.ac.uk/cmsweb/downloads/rcuk/news/UKIFAreport.pdf

BSCB Autumn Meeting 2010

5–7 September 2010. St Catherine's College, Oxford

The BSCB Autumn Meeting will be held at St Catherine's College, Oxford on the 5–7 September, 2010. This exciting meeting will be focused on Cell Organisation through the Cell Cycle and has been organized by Iain Hagan (Paterson Institute, Manchester), Gwyn Gould (Glasgow), Alison Lloyd (UCL) and Buzz Baum (UCL).Cytoskeleton and Polarity

Plenary talks

Thomas Cavalier-Smith
John Kilmartin

Growth and Form

Sophie Martin, Alison Lloyd, Jeff Errington, Keith Gull

Cell and Nuclear Division

Iain Hagan, Kathy Gould, Bill Earnshaw, Francis Barr, Kim Nasmyth

Organelles and Trafficking through the Cell Cycle

Ewald Hettama, Jodi Nunnari, Joachim Seemann, Marcos Gonzalez-Gaitan, Gwyn Gould

Cytoskeleton and Polarity

Gislene Pereira, Jordon Raff, Tom Pollard, Yukiko Yamashita, Jon Clarke

Schools News

Do you give talks in schools?

Following protests from various people including Philip Pullman and other authors of children's books, the Government, through the Home Office, has modified the 'vetting' ruling. As originally written this would have meant that virtually everyone who visited a school to give a talk or demonstration would have had to go through a 'Police Vetting' procedure.

It now appears that an occasional visit to a school to give a talk to a group will NOT require the speaker to be vetted. Certain conditions however remain in place. It is likely that this ruling will apply to the whole of the UK.

It must be remembered that as well as UK law, various conditions and rules can be made by your employer and also by the employer of the staff at the school you hope to visit. These conditions and rules can put an extra layer of protection at a local level. In many cases this appears to be to be done in an effort to protect the employer from possible litigation, but the employer has this power. This sometimes accounts for the apparent difference between what is 'allowed' in an independent school and in a nearby state school.

Recommendation: It is suggested that you ask the school that invited you to visit, to formally state and preferably in writing, their 'vetting policy' in respect of your particular visit. It is then for you to decide whether you accept these conditions. It might well then be advisable for you to inform your employer that the school has done this and that you are happy to make the visit, just in case there is any 'come back'.

David Archer. January 2010

Immune Attack

Among the many ways to expand communication between cell biologists and the public, notably school kids, computer games might seem like an unusual avenue. Inspired by movies such as Fantastic Voyage and Innerspace, the Federation of American Scientists (FAS) borrowed a page from to make an educational video game about cell biology called Immune Attack for 7th-12th graders in the U.S. This forms part of a long term project by FAS to use video games to deliver video games that can teach and train students through topics such as immune cell function. A significant aspect of this ongoing project is that the team behind it are constantly monitoring the efficacy of Immune Attack. Melanie Stegman, project leader for Immune Attack, presented some of these early findings at the American Society for Cell Biology Annual Meeting in December. Early indications are that this approach is really working in terms of student learning.

According to FAS, "American teens don't have the know-how to map a white blood cell system. Immune Attack gets them up to speed by providing them not only with text-dense screens about blood cells, but also with a full 3D game that incorporates elements of first-person shooters and Star Fox (only not in space). As a bonus, it also inspires computer studies students to make their own video games. The objective in Immune Attack is to guide your nanomachine craft through a patient's bloodstream. You're sussing out a bacterial infection, which provides you plenty of nasty things to shoot at while navigating past healthy blood cells and other naturally-occurring grossness in the human body".

More details and the download of the game can be found at http://fas.org/immuneattack/

Cell Trumps

The London based Centre of the Cell has now produced 'CELL TRUMPS' a card game along the same lines as their popular 'TOP TRUMPS'.

'CELL TRUMPS' can be ordered at the website www.centreofthecell.org at £5 per pack including VAT and post and packing. Discounts are available on 5 or more packs. There is also a special class set available for schools.

Director of the Centre Frances Balkwill says "Detailed instructions can be found on the cards and are the same as for the Centre of the Cell 'TOP TRUMPS' game. In essence the aim is to win all of the cards by picking the category with the highest score. Each card features a different type of cell and has details of how that cell 'scores' in various categories including size, number in the body and number of functions. The game is therefore useful for teaching about the multiplicity of cell types and their relationship between cell structure and function."

The Centre of the Cell opened recently and a full profile can be found in the Autumn 2009 issue of the BSCB newsletter.

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A Rough Guide to Life beyond your PhD

From nursery, infant, junior and secondary schools, A-levels and undergraduate life, the 'establishment' has imposed order and structure on both your life and the continuous process that is education. Having come this far, it's a fair bet that you have done really rather well within the system – you've worked hard, handed in on time, passed exams and now have a collection of certificates to prove it. Your ability to cram for exams, guess multiple choice answers, and rote learn facts is unquestioned.

But now, NOW everything is different. Now, as a keen lab member and future PhD, the overt trappings of timetables, fixed schedules and 'assessment' deadlines have passed. Now it's independent thought that is the key. You and your supervisor versus the bleeding edge of your chosen field of research.

No exams, no tests, no lectures.

Just hundreds of researchers publishing high power papers describing a never ending flood of scarily intelligent experiments testing fiendishly sophisticated hypotheses.

Never mind. Head down. The supervisor seems reasonably bright and must know what they are doing. After all they wouldn't have got this far if they didn't. Would they….??

Unfortunately, things start to get worse. This new world of scientific independence isn't quite the unqualified freedom it seemed at first sight. The ever-present spectre of the thesis-to-be soon rears its head and demands to be acknowledged. Soon it colours your days. Like a monster to be satiated, experiments are undertaken and figures compiled to provide fodder for the beast. Padding and tables are prepared, methods are 'written up', data comes together, and ideas take shape. After a few years the monster is looming, but at least you are getting the hang of this lab work lark. Experiments work more often than not, and you start to realise that you are beginning to think about the background to your experiments.

Then, just as you start to get good, as you start to feel that this is what being a scientist must be about, the thesis beast comes roaring back with a vengeance and time runs out. You get your head down and write and

Martin Zeidler,
University of Sheffield

slowly, agonisingly slowly the thesis takes form until finally, the tome is bound. The thesis monster has been slain. The external is invited, the viva withstood and the typos corrected.

Congratulations Doctor.

Well done! Success... Now what?

Well realistically, if you've waited this long it's probably too late. A bit of forward planning is in order and ideally 12 to 18 months before you want to start a postdoc is when to be taking serious steps towards planning for what you want to do after your PhD work.

Basically, two major options are available at this stage. To continue along the 'academic path' to do one (or maybe two) postdocs with the ultimate aim to lead your own lab, or to branch out and try something new. This is a brief rundown of a number of points that the author has come across over the years that you might find it useful to consider.

Firstly WHY:

The postdoc route is certainly appealing – you have the bench experience, you are up to speed experimentally and mentally. You are also 'in' the system, with any luck you have (or will shortly have) a paper out, you may have presented your work at meetings and you might well have contacts to other labs. In many ways, 'doing a postdoc' may well be the most straightforward option at this point. Your skills are both rare and potentially valuable to a PI and potential future postdoc supervisor.

However, this might be a good time to consider whether a postdoc really is the best route at this stage? Is this really something you have a burning desire to do, or is it the next step and path of least resistance?

Ultimately, that's a question you have to answer for yourself. And when mulling that question, it might be good to bear in mind that only a scarily tiny proportion of freshly minted PhDs end up running a lab and living the life of a high flying academic scientist. Ultimately, most PhDs (including those with a postdoc under their belts) will end up doing other things; possibly in the related areas like the biotech and pharma industries, biomedical sales, or scientific publishing. Or alternatively, many high end jobs in business and management are available both in the private and public sectors where a PhD can be a very valuable qualification. Tangible proof of motivation, intellect and independent thought.

BUT, if you think you have what it takes to run a lab, are brimming with ideas, enthusiasm and ambition then go for it. Do apply for a postdoc. And make it a good one!

Secondly MOVE:

Probably the best advice I had was to go somewhere NEW. Expose yourself to a new environment, new ideas and new challenges. If doing a postdoc is the 'easy option', then staying put is verging on lazy. And, it will not score you many brownie points with future employers. Rather take what you have learnt and experienced over the last 3–4 years and use those skills in a new environment. Build on them somewhere else. It's time to move on, preferably internationally, and expand your repertoire and continue your education.

It's also good to bear in mind that the value of movement is rewarded by funding bodies such as EMBO or HFSP which both insist on travel as a prerequisite for funding.

Moving is also good for you. Experience life. Travel. Go to a new country, learn a new language and immerse yourself in a new society. Get to know some different people and make friends. You'll keep in touch with them for years to come. This is a fantastic chance to see the world. You're young, your roots are shallow and the world is your marine bivalve.

Thirdly WHO:

This is probably the most critical aspect of doing a postdoc and the most difficult to offer concrete advice

on. Ideally you want to be able to join a happy, but productive lab, led by someone with ideas, insight and drive. Happy because life consists of more than bench work and a hostile suspicious environment is just not much fun. Productive because a lab actively generating data and publications is also likely to be at the forefront of its field, generating ideas and spin off projects that you might ultimately be able to take away with you. A publishing lab also has a higher profile and a PI likely to be giving talks, networking and reviewing – all factors that make it less likely to be taken off guard and scooped. Ultimately your boss and postdoc mentor will be absolutely KEY to your success. They will play a major role in your future both directly and indirectly. They will write your reference, direct your project and be a major intellectual and scientific influence on your life. So choose carefully!

So how to find this nirvana? Well, there are two principal ways to get a postdoc. Either reactively by applying to an advert posted in journals such as *Nature* or (more frequently these days) on the web. This will most likely be an advert for a postdoc position already awarded and linked to a specific project, a specific lab and probably a fixed time. Alternatively, approach the issue proactively by sending speculative applications to labs and PIs YOU are interested in. Both approaches have their pros and cons. An advertised postdoc position is already secure, has funding in place and the project has probably survived the rigour of a peer reviewed grant funding decision. So shouldn't be too crazy. You have a defined goal and defined timeframe to work in. This is certainly a very viable approach and is how a very large proportion of postdocs are recruited in the UK.

Alternatively a proactive approach to finding a postdoc has the potential to get you into labs not actively advertising. Hopefully, labs you are interested in are not advertising because they have reputations good enough to attract more than enough speculative applications. Applications from people good enough to apply for their own personal postdoc fellowships from agencies like the Wellcome Trust, MRC, EMBO or HFSP. Do not let this put you off. This could be YOU! If you have the CV, a publication or two and the drive then this is probably your best way to get into the top labs, especially in the US.

Fourthly WHERE:

Although the lab you are targeting is clearly the principal consideration, the environment that lab is working in will also play an important part in your postdoc. It'll have a great influence on your wider exposure to science and its reputation could be very valuable when applying for jobs in the future. You need to look at facilities and intellectual environment outside the immediate lab. Is all the stuff you will need available and accessible for you? Is there a good confocal facility? Will you be making your own fly food, bacterial media or faxing off your own orders? Or is this already provided?

For all these factors a big research institution is a plus, but at the least you want critical mass. Ideally you want to be able to interact with both other lab heads and a steady stream of high profile external speakers. Also, will you get to present your work to critical PIs outside the lab and get feedback – indeed, does anyone talk at all in the dept? Obviously, all these factors are also dependent on the WHO, and generally the best WHOs are also in the best WHEREs. But, beware - the best WHEREs are not exclusively peopled by the best WHOs.

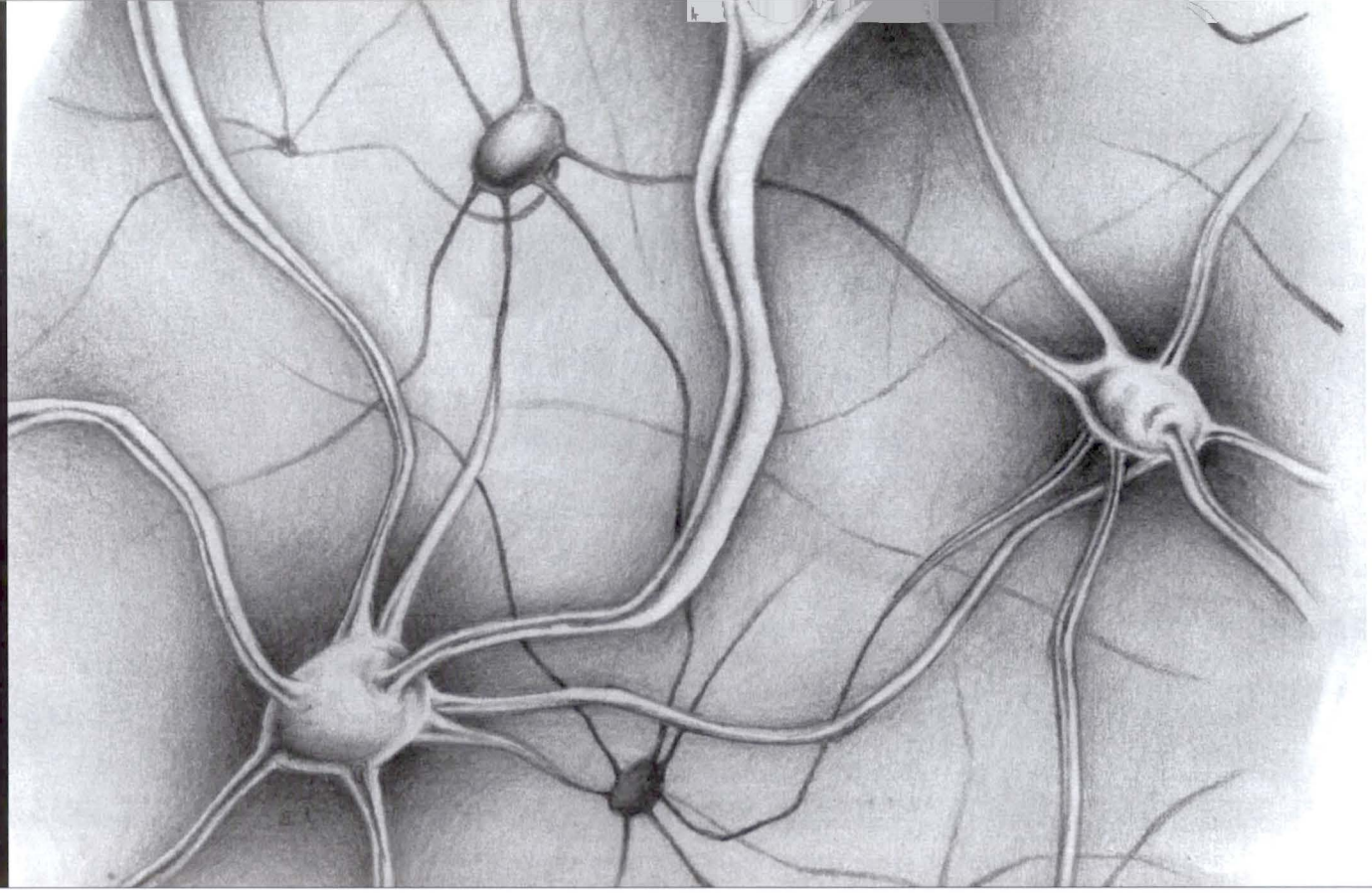
Finally, HOW:

Having identified your target labs send off an application. Make sure you highlight your scientific skills, techniques and achievements. One or more publications will come in very useful and presentations at meetings and suchlike will stand you in good stead. Prepare a good talk and make sure you introduce it well and comprehensively. Your audience will not know your work in the same way your home lab does now! Also be sure you know as much as you can about your target! Read their most recent papers. Look at their website and think about their field. Read more widely and find out who their competitor labs are likely to be. Ideally think about a project that YOU would like to do that fits into the lab as a whole. Do your homework and you are already half way there.

Once invited to visit a lab remember that this is your opportunity to shine! Do not forget that you have a rare and unusual combination of skills and would not have been invited if the PI wasn't interested. Give your talk, and if at all possible talk to the other lab members already there. These people could be your future colleagues and the source of the REAL inside story about what the boss is actually like. And finally, when you are sitting talking to a potential future boss in an interview situation stay engaged. Ask questions and make suggestions. Demonstrate independent thought and knowledge of what the lab is doing. Be yourself and be bright. And remember - an interview goes both ways. Don't forget to think about whether you can envisage this person fulfilling your needs as a postdoc: that is, your needs for a good working environment, financial stability, and above all intellectual support. Can you envisage interacting with this person for years to come? Not just over a postdoc but beyond, potentially over the course of an entire scientific career?

It's not easy, but a bit of luck and some canny choices will see you on your way. The rest, the actual WORK, is down to you. Good luck.

The author is a CRUK Senior Cancer Research Fellow and part of the MRC Centre for Developmental and Biomedical Genetics in the Department of Biomedical Sciences at the University of Sheffield. He took the advice to move very seriously and after a first degree at the University of Sussex did his PhD at the EMBL in Heidelberg. He speculatively applied to three labs in the US for postdocs and obtained both EMBO and HFSP funding for his postdoc in the Perrimon lab at Harvard Medical School. He has led his own group both in Germany and in Sheffield since 2001 and has had 10 PhD students pass through his lab in that time.



Inducing Apoptosis: Countdown to Self-Destruction

We are very pleased to announce the winner of the 2010 BSCB Science Writing Prize is Susan Turrell for her essay "Inducing Apoptosis: Countdown to Self-Destruction". The competition was judged by Vivienne Parry, freelance science journalist and regular contributor to several national newspapers and Radio 4 programmes. Vivienne commented that "it was well written, conjured up some very good images, and describes apoptosis incredibly clearly". Susan is a final year PhD student at the University of Leeds developing viral gene therapy vectors. Having graduated from the University of Sheffield with a BSc in Molecular Biology, Susan has a keen interest in the biological sciences, ranging from genetics to biochemistry and cell biology. She is especially interested in promoting science for public understanding and is looking forward to becoming more involved in this area when she has completed her research project. Susan will be awarded her prize before the Hooke Medal lecture at the 2010 Spring meeting.

From very early on in its life, a human cell is destined towards a particular fate. This job could be conducting electrical signals along a neural circuit, travelling through the body's system of blood vessels on the lookout for harmful pathogens, or sensing light that has been focussed onto the retina of the eye, allowing us to visualise the world around us. But what happens if this pre-established plan goes wrong? What if this cell becomes infected with a malicious micro-organism, or if a vital signalling pathway becomes erratic and unstable?

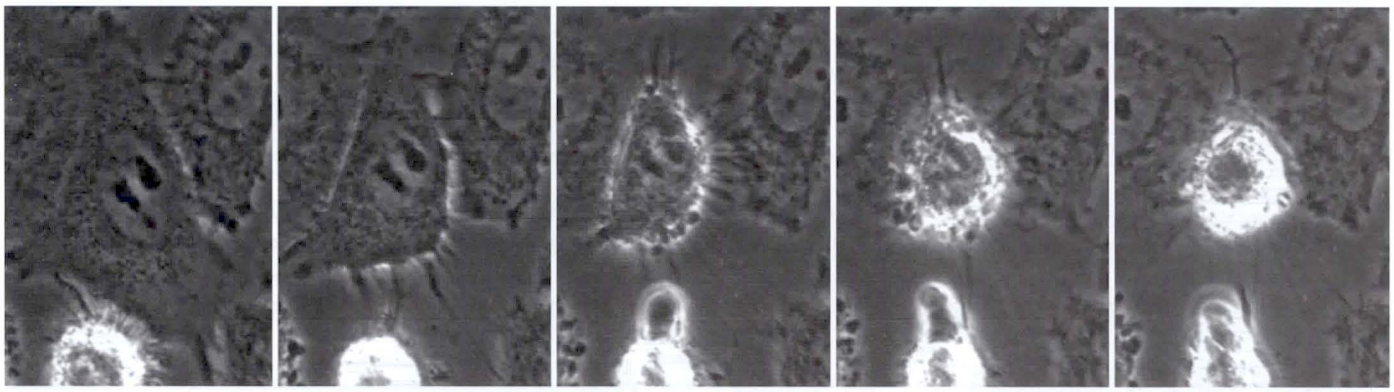
Like an ageing car, if the cell is too damaged or dangerous to mend, it's seen as a write-off and needs to be scrapped. Fortunately, every cell in our bodies has instructions for a self-destruct program maintained within its DNA. If it can't be mended, events are set in motion that culminate in the termination of that cell. This process is called apoptosis.

Apoptosis is a neat and precise method of eradicating cells in a multicellular organism. It involves the systematic shutdown of the cell, and

occurs in an ordered sequence. First, the material in the nucleus, called chromatin, condenses and the cell shrinks and contracts. Second, the nucleus disintegrates and the structures inside the cell fragment. Finally, small enveloped pieces of the cell break off in a process called blebbing. The cell has essentially been packaged up into parcels called 'apoptotic bodies' for immune cells to engulf and dispose of.

However, apoptosis is not just a method of 'clearing up' damaged cells. The cells in our bodies handover involves more and more runners. In this way the cell becomes committed to the death program and can't recover.

The final runners in this relay are a group of proteins called caspases. Caspases are expressed as inactive enzymes and have evolved to chop up other proteins when they get switched on. The termination signal is passed from 'initiator' caspases down to 'executioner' caspases, which are the bulldozers of apoptosis. These enzymes set about dismantling the structural components of the cell and this



Below: Apoptosis of human cells triggered by UV irradiation. Image courtesy of Jon Lane, University of Bristol.

are multiplying and dividing all the time, and yet we don't just keep on getting bigger and bigger. Programmed cell death balances out this growth so that the number of cells in our bodies stays relatively constant. Apoptosis is also a fundamental part of development in the foetus. It is essential for sculpting individual digits by removing the webbed tissue between our fingers and toes. It's also important in the developing nervous system. When they're growing, several nerve cells all strive to form a connection to a corresponding nerve or muscle cell. Those that make contact can transmit electrical impulses to stimulate movement or sensation, while those that fail to reach are eliminated.

So what's the mechanism behind this process? This countdown to controlled self-destruction is triggered in two ways. The cell can receive an external signal from other cells, or the process can be kick-started from within. For example, receptors on the cell surface await a signal from immune cells which are like sentries, patrolling for potentially dangerous fugitives lurking inside cells. When they recognise that a cell is harbouring pathogens such as viruses or bacteria, the immune cells release factors which cause the infected cell to commence its 'suicide program'. As well as this system, sensors inside the cell such as the protein p53 act as wardens for irreparable cell damage. p53 effectively performs an M.O.T. on the cell by inspecting the DNA contained in nucleus. Depending on the level of any damage found, it either directs the repair of the affected DNA strand, or activates the self-destruct program. It does this so that any damaged DNA is not copied and passed on when the cell divides. Once these cell sensors are activated they start a cascade that amplifies the 'death signal' so that it cannot be switched off. The signal gets passed along to different proteins like a baton in a relay race, but each protein has several batons and so each deconstruction leads to the breakdown of the cell contents.

When apoptosis stops working it can have disastrous results. One consequence is the uncontrolled growth of cells, leading to cancer. Cancer is caused by multiple mutations in different types of genes, and one of the most common proteins affected is the guardian of the genome, p53. If defective, this protein can't activate apoptosis, and therefore cells that already have damaged and mutated DNA are allowed to multiply. Lots of cancer cells also have mutations in proteins involved in the apoptotic signalling cascade, so they can grow even when the cell is instructed to commit suicide.

I'm currently developing a gene therapy vector to treat cancer. This involves transporting a gene for a protein called TRAIL into cancer cells. TRAIL recognises cells that are carcinogenic and binds to cell surface death receptors. This activates the apoptotic signalling cascade from the outside. If successful, this therapy would be specific to cancer cells, so would have fewer side effects than conventional cancer therapies. However, as some cancers have damaged apoptotic pathways, this treatment won't be useful for all cancer types. I like the idea behind this potential therapy because we are using the body's own defence system to kill the cancerous cells, we just give it a little extra ammunition.

Apoptosis is one of the mechanisms that maintains the balance between growth and stasis, health and disease. This balancing act is vital, as a problem in a tiny element of this pathway can have a massive detrimental effect. The body has evolved a way to sacrifice defective parts for the benefit of the whole organism. For this reason, each individual cell holds the seed to its own destruction.

Susan Turrell

Emmanuelle Caron: 1967-2009

The scientific community lost an outstanding researcher in 2009. Emmanuelle Caron (Manue as known by her friends and colleagues) tragically passed away on 8th July 2009 at the age of 42 following a brief battle against cancer. Manue was an excellent cell biologist and a leading scientist at the national and international levels. Manue made important contributions to the regulation of phagocytosis, the uptake of particles and microbes by immune cells. Manue had a long-standing interest in phagocytosis since her PhD obtained in Montpellier University, France, on the interaction of different bacteria pathogens and host cells.

After her PhD and with an impressive list of publications, Manue arrived in the UK for post-doctoral training in 1995: with Carlos Hormaech, University of Newcastle, for a year and then with Alan Hall at the MRC Laboratory for Molecular and Cellular Biology (MRC-LMCB), University College London. At the MRC-LMCB, She coupled her interests on phagocytosis with the emerging field of cytoskeletal regulation by the Rho family of GTPases. She published seminal papers showing that different receptors acted via distinct GTPases to mediate phagocytosis: Fc R (mediates uptake of IgG-opsonised particles) signals via Rac and Cdc42, while CR3 (mediates uptake of complement-opsonised particles) requires Rho function. These findings were intriguing and consistent with the very distinct phagocytic cups and cytoskeletal structures triggered by the different receptors (Caron and Hall, 1998). Around the attached particle, Fc R engagement promoted ruffling, a cytoskeletal event recently shown to be regulated by Rac. In contrast, CR3-dependent phagocytic cup did not contain any ruffles, but instead, the particles "sink in" the membranes, an apparent contractile event regulated by Rho. In addition, Manue also identified that another GTPase, Rap1, was necessary to activate the CR3 receptor (integrin M 2), a step essential for CR3-dependent particle engulfment (Caron et al., 2000; Self et al., 2001). These findings provided important insights into this fundamental process in innate immunity and opened up novel avenues of research.

Further collaborations allowed the dissection of downstream cytoskeletal processes driven by activation of Rho GTPases during phagocytosis. The Rho effectors ROCK and myosin II were shown to be required for CR3-dependent uptake, consistent with the activation of Rho downstream of CR3 (Olazabal et al., 2002). Interestingly, Arp2/3 complexes participated in the assembly of phagocytic cup driven by either CR3 or FcR binding, suggesting that their signalling converges at least in some regulatory aspects of cytoskeleton remodelling (May et al., 2000).

In 2002, Manue was offered a Lectureship position at the Department of Cell and Molecular Biology, Faculty of Natural Sciences, Imperial College London, where she successfully started her own lab and attracted independent funding. Her group continued to publish important contributions to signalling by phagocytic receptors in leading journals. The specificity of intracellular signalling by each phagocytic receptor was a central theme in her research, focusing on how engagement of Fc R or CR3 receptors in phagocytic cups promoted the recruitment and activation of GTPases required for efficient uptake. The latter included mapping specific regions that are essential for GTPase activation in the tail of each phagocytic receptor (Cougoule et al., 2006; Wiedemann et al., 2006). Following on from her work on Rap1 activation of the C3 receptor, her lab showed that talin, which is known to mediate integrin activation, is required for the activation of M 2 (CR3 receptor) and also plays an essential role in CR3-dependent particle uptake (Lim et al., 2007). Manue's unique expertise in studying how GTPases regulate phagocytosis was widely recognized internationally, as evidenced by the excellent reviews she wrote for top-quality journals, as well as her invitations to present her results at many conferences.

At Imperial College London, she was associated with the Centre for Molecular Microbiology and Infection (CMMI), in which she contributed extensively to a thriving community and established a number of collaborations. Manue was also instrumental in promoting cell biology at the Centre for Integrative Systems Biology at Imperial College (CISBIC). Her association with CISBIC provided a springboard to use system biology approaches to dissect and model signalling events during phagocytosis. Her old passion for pathogen/host interactions was rekindled to understand how pathogens can abrogate phagocytosis by host cells (Groves et al., 2010; Marches et al., 2008) or subversion of host intracellular signalling (Guignot et al., 2004). Similarly, her long-standing interest in cytoskeletal rearrangements triggered by phagocytosis led to modelling of how membranes engulf particles and the biophysical properties underlying this process (van Zon et al., 2009).

At both CMMI and CISBIC, Manue was very successful in attracting prestigious and long-term funding as collaborative grants at the national and European level. These achievements highlight not only the quality and impact of her research but also her unique ability to network and foster interactions across different institutions. The latter is also reflected in the number of collaborative papers in different scientific areas that Manue published consistently throughout her career.

In addition to the high productivity and top quality of her published work, Manue had a strong commitment to teaching at all levels (undergraduate and post-graduate; theoretical and practical). She was extremely gifted as a teacher and taught in many different courses at Imperial College and other Universities. The consensus from Imperial College students is unanimous: they loved her lectures, her dedication and commitment. Manue also excelled as a mentor and role model: she would personally supervise undergraduate projects in her lab and coach lab members towards further training and

career prospects.

Underpinning her success as a teacher, researcher, lab head and colleague was Manue's gift to encourage people around her to do better and excel. By example and by nurturing her lab members, graduate and undergraduate students, she enabled them to make impressive achievements. For her colleagues, Manue's brief career reminds us that it is possible to excel in research without compromising quality, mentoring and human touch.

For those that met Manue, she indeed touched each one of us in a very special way. She had a gift of putting everyone at ease around her. She was genuinely interested in knowing about each one of us and to learn more about different cultures and languages. Manue always had time to speak to you, smile and brighten up with a joke or witty comment. These small things speak volumes of the consideration and value Manue had for her colleagues and friends. Manue will be sorely missed by her family, friends and colleagues. Her untimely death leaves a large gap in our hearts and in the cell biology community. We will miss you deeply. Always.

Vania Braga[1] and Anne Ridley[2]
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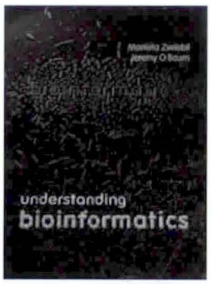
Book Reviews

Understanding Bioinformatics

MARKETA ZVELEBIL AND JEREMY O. BAUM



Understanding Bioinformatics
Marketa Zvelebil, Jeremy O. Baum
August 2007
800 pages
978-0-8153-4024-9

This book covers the wide range of topics needed to aid the understanding of bioinformatics. In the preface, the authors acknowledge the problems with writing such a book, and explain the reasoning behind the book's layout, which is set out in applications and theory chapters. This separation gives the book completeness, providing both a useful reference manual which can be quickly looked at when performing certain techniques, and also the option of reading the theory allowing a full understanding of, sometimes, very complicated concepts.

Background sections include essential biochemistry – a useful refresher – and basic information on databases and data storage options. The many sequence alignments that can be used are then covered in detail, highlighting the pitfalls and benefits of various methods, allowing the reader to make an informed decision about which is the most appropriate method for their needs. Information on the dynamic programming behind sequence alignment is also included; this is very advanced, and as a non-mathematician is at times difficult to follow.

This book provides a good source of reference for not only choosing techniques to be used but also understanding how these methods work, and as such is a valuable addition to the ever expanding field of bioinformatics. The learning outcomes highlighted at the beginning of the chapters make this a suitable text for students and recommended reading material. In addition to providing further references for suggested reading one of the particularly useful aspects of this book is that at the end of chapters it lists useful websites and search engines needed to perform bioinformatic analysis. I found this particularly useful as it provides a central resource from which to find these web addresses.

A downside is that sometimes it felt like you were just starting to get your teeth into a topic before being referred to other chapters further on in the book. Although useful to link to forthcoming chapters, this began to get a bit annoying as it sometimes felt like further discussion was needed in the section currently being read. However, as mentioned in the preface of the book the authors do acknowledge the difficulties in writing such a book and that they have tried to order the text as suitably as possible.

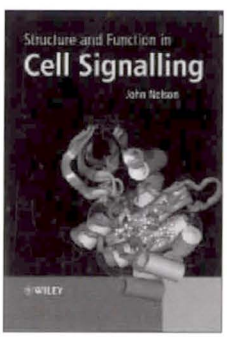
The presentation of this book is excellent. Numerous colour figures and tables break up text, and are all relevant and aid understanding. Chapters are broken down into smaller subheadings, allowing the reader to dip in and out of the book with relative ease. In addition to discussion of different alignment techniques, the book concludes with two extremely important issues essential for meaningful bioinformatic research – systems biology, and the need for data yielded from bioinformatic searches to be interpreted in terms of interactions within the larger biological system to which it belongs. The final chapter deals with the complex issue of statistics, explaining both basic statistical analysis and more advanced issues such as the need for a Bayesian approach for analysis of large numbers of genes/proteins. These statistical approaches are only touched upon and serve as an introduction to these methods, rather than a full explanation of their use.

Bioinformatics is a rapidly developing field, and as a non-expert I found this book very informative. Having read this book I now feel better equipped to plan and perform experiments in this area, being fully aware of the problems to look out for and the strengths and weaknesses of the programs and software available for this research. I would recommend this book to anyone wanting to investigate or perform bioinformatic research as I think this text forms a solid basis on which to learn both applications and theory behind bioinformatics for the beginner, and will also serve as a good reference text for those with a more advanced knowledge of the area.

Paula Williams
University of Nottingham

Structure and Function in Cell Signalling

JOHN NELSON



Structure and Function in Cell Signalling
John Nelson
Pub: Wiley Blackwell
ISBN: 0470025514
410 pp
July 2008

In this book, John Nelson invites us into the world of molecular structure and function of signalling molecules in cells, a field often ignored by publishers. His main focus is on protein domains and modules involved in intracellular signalling cascades, the functions of which are explained in great detail. Post-translational modifications of proteins (and their domains) are essential elements of signalling in all living cells. Their effect on function of signalling molecules and their structure is explained with special care.

These chapters dominate the text, are accompanied with elegant ribbon-based models of proteins and their interacting partners, and present a comprehensive and valuable tool for teaching on how cells recognize, transduce and respond to environmental stimuli. The author underlines the importance of small molecules in signalling processes, including lipid molecules of cellular membranes, due to their different physical properties complementing proteins in their capability to respond to a wide range of stimuli in the highly noisy environments of extracellular space and cellular cytoplasm. Some pathways are described in much detail in the final chapters to illustrate the complexity and modularity of signalling processes as we can describe them today.

The Appendix includes a brief but comprehensive explanation on how RasMol, an open-source software available online, can be installed on computers and used for viewing and analysis of protein structures, domains and motifs.

A reader will be pleased by a lot of useful information which is not available in any individual publication elsewhere. Unfortunately, this book is missing the lightness of current textbooks that is appreciated especially by younger readers. The text is poorly structured, editing is poor and the quality of graphics brings us back in time by at least 10-15 years. Some details describing specific information on individual molecules (probably favoured by the author) are presented repeatedly throughout the text without an obvious reason. At the same time, numerous basic terms and principles of cell signalling are missing or explained poorly. The book would benefit from the addition of introductory sections describing general principles and characteristic molecules responsible for signalling in living cells, written using a more popular and educational style. This would increase the interest of more readers for the later more technical and specialised sections focused on structural motifs/modules and molecular mechanisms driving the processes of cell signalling with great precision.

Finally, the book is almost exclusively focused on the information obtained from *in vitro* experiments. Discussion of structural and functional data from *in vivo* imaging techniques would bring to life the processes described in this book. Also, such a text could then be more appreciated by readers focused primarily on cell biology or clinical science.

Marek Cebecauer
Imperial College London

Meeting Reports

American Society for Cell Biology 2009 Annual Meeting

5–9 December 2009. San Diego, CA

A meeting report about the ASCB conference in San Diego, but where to start? So many impressions, so many talks and so many posters! First, a big compliment to the organisers for the great organisation, the huge variety of topics presented in talks or posters, the abstract book, which helped me a lot to organise my days through out the meeting, and the helpful people everywhere around in the convention centre who could answer questions.

The ASCB conference was a great opportunity to hear about the latest advances in science. I enjoyed listening to the keynote lecture "Stem Cells, Pluripotency and Nuclear Reprogramming" of Rudolph Jaenisch, as well as the large choice of talks during the morning sessions. Highlights were the sessions on cell polarity and intracellular trafficking on Sunday with talks addressing the interaction of the exocyst, Rab8 and Rab11 being very close to my project. A point, which made the conference especially interesting for me, was that the majority of labs working in the field of my Ph.D project, the exocyst and rab proteins, are based in the USA. I met many people whom I knew from publications for the first time, and we had interesting discussions at the posters. I appreciated that people were open and motivated to share their results. In addition, interesting input was provided on open questions of the project. For young scientists, these poster sessions can be the most fruitful part of the conference.

As I went to the ASCB conference between my PhD and my first postdoc position, I had the opportunity to find out about being a postdoc, to meet people working in that field and to introduce myself. Networking was therefore an important part of the conference. Furthermore, San Diego was a great place to visit, and conference an event which was really worth being part of.

Miriam Essid
Institut Pasteur, Paris

International Society for Stem Cell Research (ISSCR) 7th annual meeting

8–11 July 2009. Barcelona, Spain

The International Society for Stem Cell Research (ISSCR) annual meeting is one of the finest international stem cell meetings, attracting more than 2,500 of the leading stem cell professionals from over 50 countries around the globe. The fact that this year the meeting was held in Barcelona, was just icing on the cake!

The venue was the impressive Centre Convencions Internacional Barcelona (CCBI), located just 5 minutes off the Spanish coast. This is a purpose built establishment, featuring numerous conference rooms, exhibition halls and press rooms, spanning over four floors.

The meeting was co-sponsored by Boston University, and the Spanish Centres of Genomic Regulation and Regenerative Medicine.

The keynote lecture was delivered by Prof. Nancy Wexler (Columbia University), who impressed everyone with the moving story of her quest to gather a pedigree of over 18,000 individuals from Venezuela that helped identify the gene responsible for Huntington's disease in humans. Prof. Jane Rossant (Hospital for Sick Children, Canada) gave The Anne McLaren Memorial Lecture on "Making stem cells and establishing cell fate in the blastocyst", while Dr. Olivier Pourquie gave the European Molecular Biology Organisation (EMBO) plenary lecture on "Spatio-temporal compartmentalisation of metabolic programmes during muscle precursor differentiation".

As six out of the eight plenary sessions of the conference were focused on topics very relevant to my PhD (embryonic stem cells and induced pluripotency stem cells) the ISSCR greatly broadened my overall knowledge of current research and opened-up my eyes to the plethora of institutes around the world that carry out stem cell-related work, a lot of which were advertising available post-doctoral positions. It was also delightful to attend talks by some of the most respected scientists in the field of my PhD studies, such as Prof. Shinya Yamanaka (Kyoto University, Japan) and Dr. Kathrin Plath (UCLA, USA) who delivered talks on the mechanisms of iPS cell formation and characterisation.

The poster I was presenting on "Epigenetic insights into the formation of human iPS cells" attracted a lot of attention on all three poster-display sessions. Young scientists as well as principal investigators became interested in the work we carry out here at the University of Nottingham. Both the talks and poster-sessions were very inspirational and gave me good ideas for experiments that I could perform to complete my thesis. Also, during the course of the conference I never missed a chance to advertise the 5 post-doctoral positions that were available within our lab at the time, as well as the Masters course on "Stem Cell Technologies" that our department has been running for the past two years. My supervisors made sure that I was equipped with flyers which I handed out generously!

The ISSCR meeting was not short of social events! Spanning though a period of four days, it gave us a chance to explore Barcelona by night, which can be magical over the warm summer month of July. Both the conference organised dinner and cocktail party at the Royal Palace of Pedralbes, and our visits to the bars of the cosmopolitan Barcelonetta helped make my stay memorable.

Many thanks should go to my sponsors – The British Society for Cell Biology, The British Society for Developmental Biology and the University of Nottingham's Graduate School – who provided funding that allowed me to attend this event.

Elena Matsa
Wolfson Centre for Stem Cells, Regenerative Medicine and Mathematical Modelling (STEM)
The University of Nottingham

In the beautiful Barcelona, Spain, more than 3,100 researchers from all over the world get together to present their cutting edge research in stem cell sciences.

The meeting has seven plenary sessions covering broad areas of embryonic and adult stem cell biology, induced pluripotent stem cells (iPSCs), cancer stem cells, developmental biology, regenerative medicine and translational research. More than 100 talks and 1700 posters were presented during the course of 4 days. It is impossible to cover all the sessions and topics, so I will only report some highlights and exiting progress presented in the meeting.

In the opening talk about brain regeneration and disease repair, Nancy Wexler, told the story that how she and her colleagues discovered the mutated gene underlying Huntingdon disease in remote villages of Venezuela and emphasised the reason why there are so many expectations on stem cell research – to treat devastating disease.

Marianne Bronner-Fraser talked about her new findings on the neural crest cell formation and migration during embryogenesis. JmjD2A binds and demythylates histone H3K9me3 mark on neural crest marker gene *Sox10* to regulates its level of expression. By analysing putative transcriptions sites of *Sox10* promoter, her lab showed that Sox9, cMyb and Ets1 directly activate *Sox10*.

Fred Gage gave an interesting talk about unusual high frequency of L1 retrotransposition in human brain compared to other tissues. L1 insertion can change DNA sequence and cell properties. This finding suggests that the neuronal cells in one's brain may contain difference DNAs, which contribute to the extraordinary capability of human brain.

In plenary session II, signals controlling renewal and differentiation, Lin Haifan from Yale University, USA, presented data implying that translation regulation is a novol mechanism defining the stem cell fate. MILI and MIWI, members of PIWI/Argonaute protein family are enriched in germline cells. Disruption of mili in mice did not affect mRNA level in the developing spermatocytes but reduced the rate of protein synthesis, the affected germ cells failed to self-renew or differentiate.

Azim Surani from the Wellcome Trust Gurdon Institute, Cambridge, UK talked about interesting findings that a small number of mouse epiblast stem cells can revert to ES cells and reset their epigenetic

state after switching culture condition.

In the plenary session V, Growth control in Stem Cells and Cancer, Thea Tlsty gave a nice talk about the epigenetic plasticity in stem cells and tumor cells. She showed evidence that hypermethylation of the p16 promoter may be an indication of pre-clonal phase tumorigenesis in epithelial cells. This accompanied with up-regulation of PcG proteins EZH2 and SUZ. These could be important biomarkers of early carcinogenesis.

In the afternoon session about differentiation of stem cells, Deng Hongkui from Peking University, China, talked about how to direct differentiate of human ES cells and iPSCs into functional hepatic cells and hepatic progenitor cells that can become either hepatocyte-like cells or cholangiocyte-like cells.

iPSC is an ultra fast growing field. There are more than 300 posters about iPSC. Several posters described using novel reagents such as small molecules or recombinant proteins to induce pluripotency. Hopefully in the near future, scientists can make iPSCs without using DNA.

Konrad Hochedlinger, assistant professor, Department of Medicine, Cancer Center and Regenerative Medicine Massachusetts General Hospital, USA, received the Inaugural ISSCR Outstanding Young Investigator Award, for his work on cloning and cellular reprogramming. He delivered an exciting talk about creating an inducible system to make human iPSCs and use it to perform genetic and chemical screens to identify new molecules important for inducing pluripotency. iPSC pioneer, Shinya Yamanaka also gave a talk on their work to generate virus free iPSCs.

In the session on epigenetics and reprogramming, Joel Blanchard from Harvard Stem Cell Institute, USA, showed that reprogramming factor Sox2 can be replaced by a small molecule that inhibiting TGF signalling. Li Han from Spanish National Cancer Research Center and Juan Carlos Izpisua Belmonte demonstrated that Ink4/Arf tumor suppressor locus and p53 pathway are barriers for reprogramming. Disabling these "roadblocks" significantly increases reprogramming efficiency but also makes cells susceptible to carcinogenesis.

I enjoyed the Barcelona ISSCR meeting a lot and would like to thank the BSCB for the Honor Fell Travel Award that enable me to attend this important meeting.

Jie Na
Department of Biomedical Science,
University of Sheffield

The ISSCR annual meetings provided an opportunity to stem cell scientists to present their work. This year's meeting, which was the first to be held in Europe, was very successful attracting more than 3100 scientists from all over the world. The meeting was co-sponsored by the Centre for Genomic Regulation (CRG) and the Center of Regenerative Medicine in Barcelona (CMRB).

The program, divided into seven plenary and three concurrent sessions, included a diverse range of topics such as Brain Regeneration and Disease Repair, Signals Controlling Renewal & Differentiation, Regulating Cell Type in the Endoderm, Hematopoietic Stem Cells in Differentiation & Disease, Growth Control in Stem Cells and Cancer, Stem Cells as Model Systems and Reprogramming and Epigenetics.

The meeting opened on Wednesday 8th July, with plenary session I focused on Brain Regeneration and Disease Repair. The keynote lecture was delivered by Nancy Wexler (Columbia University, The Hereditary Disease Foundation, USA), who talked about her discoveries of the genetic mutation responsible of Huntington's disease.

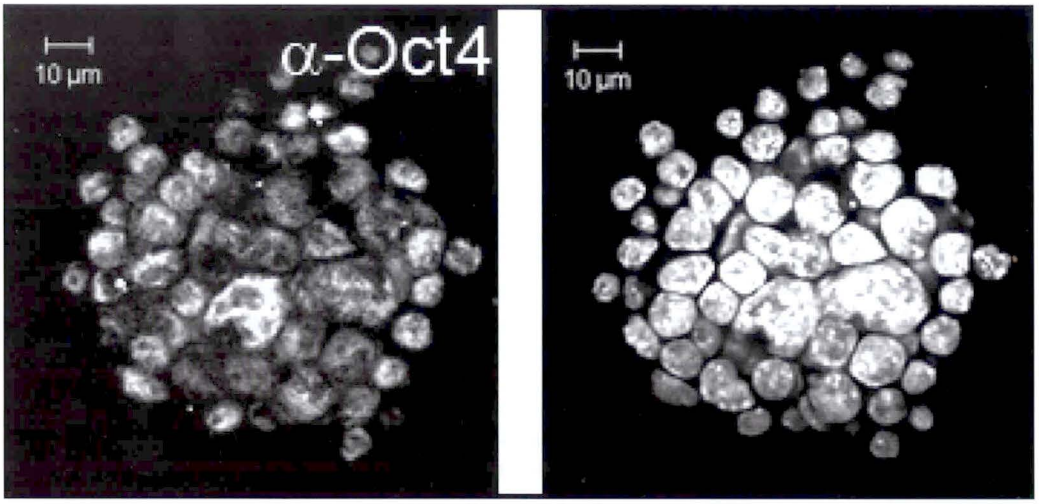
The second plenary session, also held on Wednesday, was based on signals controlling renewal and differentiation. In this session, Lin Haifan (Yale University, School of Medicine, USA) gave an interesting talk about a novel mechanism of translational regulation involved in maintaining stem cell identity. He presented data involving two protein members of the PIWI/ARGONAUTE protein family, MILI and MIWI as positive regulators of translation. The second plenary session closed with the presentation of the Inaugural ISSCR Outstanding Young Investigator Award, supported by the University of Pittsburgh. The award, presented by Rudolf Jaenisch (Whitehead Institute for Biomedical Research, USA), went to Konrad Hochedlinger, PhD, (Massachusetts General Hospital, Boston, MA USA) for his exceptional achievement in stem cell research.

The first day closed with an all-attendee mixer, supported by the Government of Catalonia, in the Royal Palace on Pedralbes. This event was an excellent opportunity for meeting other scientists, and to present and discuss research in a relaxing and friendly atmosphere.

The second day, Thursday 9th July, opened with a plenary session in Regulating Cell Type in the Endoderm. Sarah Ferber (University School of Medicine, Miami, USA) delivered the most fascinating talk of the session. She presented data showing that functional endocrine pancreas can be generated from adult human liver by ectopically expressing transcription factors such as Pdx1. Sarah highlighted the benefits of using autologous cell replacement to minimise the risk of immune rejection and to overcome the limited supply of tissue for transplantations.

The plenary session was followed by session I consisting of 5 concurrent sessions with the following topics: Embryonic Stem Cells, Stem Cells in Model Organisms, The Ethics of Egg Sharing, Cell Cycle and Timing Mechanisms and Controlling Tissue Stem Cells. Michael Edel (Centro de Medicina Regenerativa de Barcelona CMRB, Barcelona, Spain) explained the importance of Rem2 GTPase in regulating human embryonic stem cell (hESC) cell cycle and apoptosis. Moreover, he presented evidence suggesting that Rem2 increases reprogramming of human somatic cells to induced pluripotent stem (iPS) cells by regulating genes involved in cell cycle.The last session of the day, Plenary IV, discussed hematopoietic stem cells in differentiation and disease.

The poster exhibition started on 9th July, and was divided in two sessions (Thursday and Friday) as the number of posters presented exceeded an astonishing 1700. Posters were in displayed for the duration of the meeting. During Thrusday's poster session, Grassi Gonzalez (CMRB, Barcelona, Spain) presented very interesting work based on the generation of mouse iPS cells from mouse embryonic fibroblast by using a non-viral polycistronic vector expressing Oct4, Klf4, c-Myc and Sox2. He also highlighted the clinical relevance of



this work as iPS cells without transgene integration are safer for clinical use than those where the vector has integrated into the genome. Another fascinating work in the field of generating iPS cells was presented by Marcia Riboldi (Fundacion IVI, Valencia, Spain). She showed that treatment of mesenchymal stem cells, isolated from Amniotic fluid stem cells (AFS), with chromatin remodelling agents such as TSA or 5-Aza leads to generation of hESC-like colonies. This work has a great clinical significance also as it does not involved the introduction of foreign DNA.

The final event of the day was the Junior Investigator Social Hour, which was supported by Pfizer/Regenerative Medicine and took place in the Sagrada Familia Room at the AC Barcelona Hotel. This was again a great opportunity for networking and discussing work with peers.

The third day of the meeting, Friday 10th July, started with a Plenary Session on Growth Control in Stem Cells and Cancer. Thea Tlsty (University of California, San Francisco, USA) gave a very interesting talk about how cancer could be detected, and therefore treated, at early stages by identifying early epigenetic changes. Thea explained that epithelial cells, which are not yet carcinogenic, that have hypermethylation of the p16 promoter are more prone to become tumorigenic. She also talked about other epigenetic changes which lead to pre-clonal phase of tumorigenesis.

The next event of the day was concurrent session II, divided again in five different topics: Differentiation of Stem Cells, Stem cell Fate Choice, Stem Cell Lineages, Asymmetry and Specification, Stem Cell Technologies, and Tissue Specific Stem Cells. In the Stem Cell Fate Choice session, Stuart Orkin (Harvard Medical School Dana-Farber Cancer Institute, Boston, USA) presented data highlighting the importance of the Polycomb complex in regulating self-renewal and differentiation in embryonic stem cells. Another interesting talk during this session was given by Jonathan R. Yeh (Department of Medicine, McGill University, Montreal, Canada). He showed evidence that the canonical Wnt pathway does not support self-renewal of Spermatogonial Sperm Cells (SSCs) but addition of recombinant Wnt5a maintains SSCs *in vitro* and inhibits the canonical pathway.

Following this session, a Meet the Experts Lunch session was organised where attendees had the opportunity of meeting some of the speakers and discussing research in a friendly and relaxed environment. I had the pleasure to have lunch with Maria Blasco (Spanish National Cancer Centre (CNIO), Madrid, Spain) who explained different research lines that are currently ongoing in her lab. One interesting area of research in her lab investigates the interplay between DNA repair signalling and telomeres. She also explained about research in her lab that aimed to investigate which nationality in Europe has the longest temolere and whether this is related to life expectancy. The study indicated that the French have the longest telomere, which correlated with the highest life expectancy.

The final session of the day before poster presentations was Plenary Session VI and the topic was Cells that Build Bodies. In this session, Olivier Pourquie (Howard Hughes Medical Institute, Kansas City, USA) delivered the EMBO Lecture, which was supported by EMBO. He explained their findings of how cellular physiological programs are spatially and temporally compartmentalised during differentiation of muscle precursors. The day finished with the second poster presentation.

The last day of the conference, Saturday 11th July, started with VII plenary lecture in Stem Cells as Model Systems. The most fascinating seminar of this session was given by Shinya Yamanaka (Kyoto University, Japan) who explained how, first in mouse, and then in human, pluripotent stem cells were induced in his lab by introducing Oct4, Sox2, C-Myc and Klf4 into fibroblast through retroviral infection for the first time ever. Moreover, he also talked about the development of safer iPS cells by removing C-Myc, and using plasmid vectors instead of retrovirus. Another remarkable talk was delivered by Konrad Hochedlinger (Harvard University, Boston, USA), who discussed different aspects surrounding the generation of iPS cells. Among those were ways to improve efficiency of reprogramming by using drug-inducible versions of the reprogramming factors, and how to make iPS cells safer by using adenovirus instead of retrovirus.

Next event of the day was the final Concurrent Session III again consisting of five tracks: Stem Cells and Cancer, Epigenetics and Reprogramming, Systems Biology and "omics" of Stem Cells, Clinical Translation of Stem Cells and Stem Cell Niche.

The closing session of the conference was about Reprogramming and Epigenetics. Juan Carlos Izpisua Belmonte (Center for Regenerative Medicine, Barcelona, Spain) presented data suggesting that reprogramming efficiency of somatic cells can be considerably increased by inactivating p53. The meeting closed with The Anne McLaren Memorial Lecture given by Janet Rossant (Hospital for Sick Children, Toronto, Canada).

I am very thankful to the BSCB for awarding me the Honour Fell Travel Award that allowed me to attend to this exciting meeting.

Yolanda Sanchez Ripoll
Centre for Regenerative Medicine
University of Bath

Society for Developmental Biology 68th Annual Meeting

23–27 July 2009. San Francisco

The 68th meeting of the Society for Developmental Biology (SDB) took place in the Hyatt Regency hotel in foggy San Francisco, California.

The meeting started with an impressive line-up of plenary speakers. Elliot Meyerowitz (Caltech) opened the meeting by advocating the importance of combining model building, live imaging and experimental evidence in addressing developmental questions. Meyerowitz presented evidence that plant meristem cells can feel stress from adjacent cells and respond to this stress internally. In turn this physical stress is important for morphogenesis. Claudio Stern (UCL) also supported the importance of model building in his talk "Gastrulation: From cells to embryo." Nadia Rosenthal (EMBL, Italy) closed the session with an interesting talk about enhancing mammalian regeneration, in which she addressed the question why have mammals lost their regenerative capacity? Rosenthal's work on the regenerative capacity of the mouse heart showed that the Notch pathway enhances cardiac recovery.

I spent the first morning of the conference in the "Developmental Neurobiology" symposium. David Wilkinson (MRC-NIMR) presented his work on the generation of non-neurogenic zones in the hindbrain. Progenitor cells in the centre of rhombomeres are maintained by Fgf signalling. He showed that Fgf20a, expressed by neurons located in the mantle zone, signals back to the progenitors and inhibits neurogenesis, thus maintaining non-neurogenic zones. Ben Novitch (UCLA, Los Angeles) also addressed the question of how progenitor cells are maintained, more specifically how Olig2 progenitors in the spinal cord are maintained to give rise to two distinct neural lineages, more specifically motor neurons and oligodendrocytes.

The next session gave postdocs the opportunity to present their work, including me. I have to admit that I was very nervous before giving my talk and as a result processed little of the talks that went before mine! However, all the talks were of a very high standard and I was honoured to be amongst such high-calibre speakers.

2009 marked two special anniversaries for the Society of Developmental Biology; the Society itself was 70 years old, and the journal Developmental Biology had reached its half-century. As these anniversaries coincided with Darwin's birthday, there was a strong evolutionary developmental biology theme to the meeting. This was evident in the plenary sessions. The first was entitled "Evolution of Developmental Regulatory Systems". Patrick Lemaire (IBDML, Marseille, France) talked about the very different developmental programs of ascidians versus vertebrates. Comparing ciona and zebrafish, he showed that local chromatin cues differ between compact and large genomes and this resulted in the very different developmental programs. He hypothesised that the nucleosome occupancy landscape may be differently regulated in compact versus large genomes and this may also be true for fast versus slow developing organisms. Edith Heard (Institut Veronique Duranthon, Paris, France) talked about the different ways that mammals inactivate the X chromosome during early development. The imprinted form of X inactivation is the ancestral mechanism whereas random X inactivation is the more recent form. In mouse, both mechanisms are employed, and Heard showed how studying early imprinted inactivation might uncover its evolutionary origins. The session was ended by Alejandro Sanchez Alvarado (HHMI, Utah) who showed us some amazing movies of planarians with two heads or two tails at each end of the body axis. He demonstrated that altering the levels of B-catenin determined whether the planarians developed two heads or two tails.

On Saturday morning, Sean Morrison (Uni. Of Michigan) kicked off the "Neural and tissue specific stem cells" symposium. He talked about some of the novel regulators of mammalian stem cell self-renewal identified by a recent forward genetic screen carried out in his lab. In particular he showed some of his data that demonstrated that Prdm16 is expressed in human stem cells, and is also required for maintenance of normal stem cell frequencies in the brain. Nathan Mundell (Vanderbilt University, Nashville) also talked about a gene required in the maintenance of stem cell characteristics in neural crest (NC) cells. Foxd3 is one of the earliest markers of NC and Mundell demonstrated that it is required for self-renewal and multipotency. Neurospheres derived from Foxd3 deficient NC cells are reduced in size and give rise to fewer secondary spheres. Additionally, mutant NC cells precociously differentiate in the absence of Foxd3. After coffee I switched to the "Pattern Formation" session, which included talks from Richard Harland (Berkeley) and Igor Dawid (NIH, Bethesda). The session also included a talk from Malcolm Maden (University of Florida) who introduced us to some new players in the retinoic acid signalling pathway, including a receptor, Stra6.

The evening plenary session, "History of Developmental Biology" was a special session, again in keeping with the anniversary celebrations. This session included interesting talks from Diana Buchwald (Caltech), William Friedman (University of Colorado), Manfred Laubichler (ASU, Arizona), Susan Ernst (Tufts University, Massachusetts), Claudio Stern (UCL) and Jane Maienschein (ASU, Arizona). Several of the talks focussed on Charles Darwin, however we also heard about Erasmus Darwin (Charles' grandfather) who was also a biologist and had proposed the idea of a single common ancestor. We also learnt about Hofmeister, a botanist who wrote love poems about gingko nuts! Claudio Stern

(UCL) gave a talk about Waddington, who despite the massive impact he made on the developmental biology field, did not devote his life to the field, but was actually an expert on almost everything it seems.

I spent Sunday morning in the "Epigenetic influences on Development" session. Christine Reid (University of Pennsylvania, USA) described how the Wnt and Nodal pathways interact during the formation of the organiser in Xenopus. The Nodal pathway upregulates FoxH1, whereas Wnt upregulates Siamois/Twin, which in turn both bind to Goosecoid and induce its transcription. The organiser is formed where Wnt and Nodal expression overlaps, and the interaction of the two pathways induces the expression of Goosecoid. Further work from Reid showed that the common co-activator was a Histone acetyltransferase (HAT) p300. Roberto Mayor (UCL) presented his labs work on NC cell migration. He showed that NC cells undergo a directional migration as a result of a combination of co-attraction and contact inhibition, rather than utilising a chemoattractant/repellent. Mayor again demonstrated the importance of computational programs to model the behaviour of cells. Finally in this session, Michael Harrison (MRC Centre for Developmental and Biomedical Genetics, University of Sheffield) presented his work on the role of Hdac1 in neurogenesis in the zebrafish embryo. Harrison proposed that as well as a role in silencing genes; Hdac1 may also promote transcription, specifically of proneural genes.

The final session of the meeting was, as tradition dictates, the award session. The Edwin G Conklin medal was presented to David Kingsley (Stanford), and the DB-SDB Lifetime achievement award went to Donald Brown (Carnegie Institute for Science). Sean Carroll (University of Wisconsin-Madison) won the Viktor Hamburger Outstanding Educator award, and should also have won best talk prize, if such a prize existed! In his typically enthusiastic and enthralling style, Carroll presented to us the story of Louis and Mary Leakey. He then brought the meeting to a close with an amazing 'Happy Birthday' video for Darwin.

I would like to thank the BSCB for providing me with an Honor Fell travel grant, which enabled me to travel to this fantastic meeting.

Caroline A Pearson
MRC Centre for Developmental and Biomedical Genetics and Department of Biomedical Science,
University of Sheffield

The 68th annual meeting for Society for Development Biology was held at the luxury Hyatt Regency near the bay in San Francisco. San Francisco is one of the most unique cities in the world; the beautiful streets, wonderful weather and ingenious transportation system only served to increase our enjoyment of this amazing city. California is a leading centre of academic creativity and at the forefront of biotechnological research and we were honoured to be a part of it.

The five day conference included more than one hundred talks, divided into nine sessions and around five hundred posters presented in two poster sessions every evening. Prior to the meeting, two satellite symposia titled "Neural crest and Ectodermal placodes" and "Plant development in the changing world" were presented. Most interestingly, they provided the "Historical session", presenting several major milestones in developmental biology. The conference was all encompassing, covering all aspects of developmental Biology as well as several new experimental techniques and cutting edge research. Groups had travelled from all over the world from places such as Japan, Asia, Europe and the UK bringing new perspectives and ideas to the meeting.

From individual cells to an embryo, and the embryo to the adult body, the study of development has provided great insights into phenotypic evolution throughout the 19th and early 20th century. This year, many events have taken place to commemorate the 200 years since the great naturalist Darwin was born and the 150 since the publication of his book, The Origin of Species. However, these were not the only significant anniversaries in 2009 and we were delighted to be able to attend the 68th SDB conference that celebrated the 70th anniversary of the Society for Developmental Biology and the 50th anniversary of the journal Developmental Biology.

This conference covered a wide variety of sessions relating to developmental biology. There were more interesting talks than I have room to describe here; however the talks given by Claudio Stern (University College, London) really drew my attention. His first talk covered the history of gastrulation, from the cellular level to the whole embryo. He reviewed in detail gastrulation in different groups of organisms, such as *Xenopus*, chick and mouse. In addition, he described the many different cell movements and activities that result in symmetry breaking in the embryo, the establishment of the body axes, and the induction of the germ layers.

The second talk he gave gave an overview of the contributions of Conrad Hal Waddington (1905–1975) to developmental biology. Waddington's early desire was to become a palaeontologist, however inspired by the discovery of the primary organizer by Spemann and Mangold in 1924, he diverted his interests to genetics and experimental embryology. He developed the concepts of evocation and individuation, which represented the evocator, a morphogenetic stimulus, and the organizing effect, the consequence of induction. In addition, concepts such as epigenetic interactions and changes in the regulation of zygotic genes can produce phenotypic change in development and evolution are a direct legacy of Waddington's conceptualization of the integration of genetics, development and evolution through epigenetics. Such overview talks are extremely beneficial and remind us of the context in which our daily work is based.

Alongside the talks about the history of developmental biology, I would like to highlight a stunning talk presented by Josh Sanes

(Harvard University, Cambridge). Josh introduced the Brainbow mouse, a transgenic mouse that provides a new way to map the brain's neuronal circuitry. Rather than using direct staining to see nerve cells and their connections, Josh and co-workers demonstrated that they can insert genes coding for different-colored fluorescent markers into transgenic mice and express pure red, yellow or blue fluorescence in individual neurons. However, not satisfied with a single pure color, and in order to be able to distinguish more cells in one mouse, they created a DNA recombination system capable of generating a vast range of colours. By clever combination of a transgenic mouse carrying three or more distinct fluorescent proteins and the cre/lox recombination system, random splicing events resulted in each neuron expressing a different subset of the fluorescent proteins. The Brainbow mouse provides a useful tool which will allow us to map glial territories and neurons *in vivo* and visually study the neuronal circuitry.

Alongside listening to other scientists present their work, I (Yi-Hsien Chen) presented a poster on the roles of Nodal and Mix-like genes in mesoderm induction in a urodele amphibian, the axolotl. I received lots of helpful feedback and insight from many outstanding scientists. Overall, this conference provided me an opportunity to interact with world-leading scientists and also served to further my knowledge of developmental biology.

Yu-Huan Shih also presented his poster about the function of Sox3 in regulation of formation of the Neiuwkoop centre in zebrafish in the second poster session on fourth day. His results demonstrated the importance of Sox3 throughout the early embryo, suggesting that Sox3 is a primary determinant of the earliest pattern formation in zebrafish. He was so glad to meet so many brilliant scientists in the same field, providing so many precious advices and helping my research and thesis more completely.

In the final evening, we had formal Banquet and then danced with live band in the Grand ballroom in the hotel. Overall, the conference offered a fantastic opportunity to meet scientists from the entire world and share valuable experience together. We definitely recommend future SDB meetings for everyone in developmental biology field. We would like to thank to the BSCB for providing this Honor Fell Travel award which allowed us to attend this amazing conference on the other side of America.

Yi-Hsien Chen and Yu-Huan Shih
Institute of Genetics, Queen's Medical Centre, University of Nottingham

International Gap Junction Conference 2009

25-30 July 2009, Sedona, Arizona, USA.

Every two years, researchers from diverse fields in cell biology, physiology and pharmacology meet at the International Gap Junction Conference to discuss their findings.

Gap junctions are clusters of protein channels that allow cells to freely exchange small molecules and ions. Each channel consists of two hemichannels in each membrane of the adjacent cell, and each hemichannel consists of six subunits of connexin protein (Cx). Research in gap junctions has gained great importance over recent years, as mutations in connexin genes and modification of gap junction quantity and connexin expression play a key role in human diseases such as cardiac arrhythmias, congenital deafness, oculodentodigital dysplasia or keratodermas.

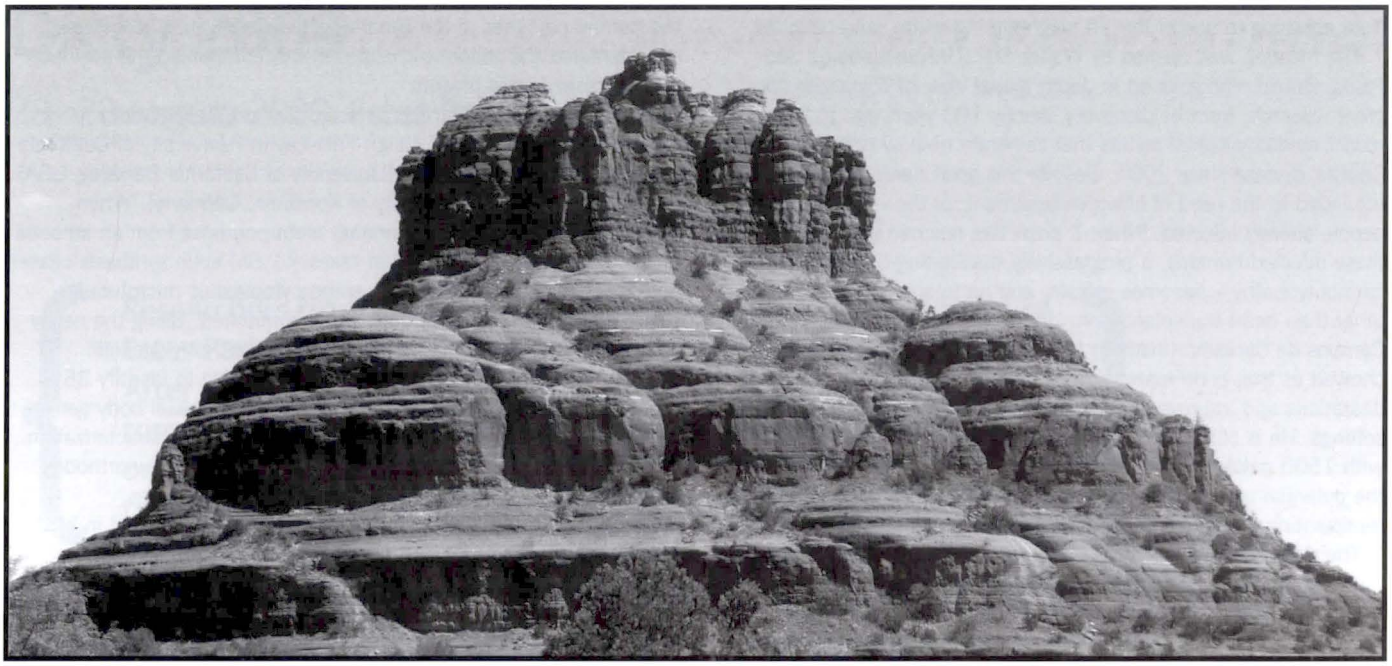
The conference hotel was located between stunning red sandstone formations in the desert, which we could explore on two mornings designated for free time. On Saturday night, a reception was held in the Hilton hotel with cake and drinks to welcome researchers from all over the world. The lectures officially started the next morning (Sunday) and the first session dealt with connexins and their functions in different tissues. Keynote speaker Paolo Meda (University of Geneva) focused on the role of connexin signalling in diabetes. Beta cells that do not express Cx36 fail to communicate correctly which interferes with their stimulation and eventually the release of insulin is altered. Connexins play a role in many tissues; other researchers are working on the role of connexins in wound healing of the skin (Paul Lampe, Fred Hutchinson Cancer Research Centre, Seattle), in the lens of the eye (Jialu Liu, University of Texas) and the retina of Zebrafish (John O'Brian, University of Texas). During the conference there were entire sessions dealing with connexins in the heart, the vasculature and the nervous system. Eliana Scemes (Albert Einstein College of Medicine, New York) presented data that demonstrate that the C-terminus of the Cx43 protein is important for signalling mechanisms involved in the development of neurons in the embryonic brain. A very interesting presentation was given by Colin Green (University of Auckland, New Zealand). He showed that four patients with severe injuries of the cornea recovered faster when antisense oligonucleotides that down-regulate the expression of Cx43 were administered. This was due to improved corneal epithelial repair. His co-worker Jie Zhang (University of Auckland, New Zealand) applied similar methods to study repair of the spinal cord after injury. She showed that reducing the level of Cx43 prevents the spread of the injury and reduces the formation of scar tissue.

Whereas many groups work on the role of connexins in tissues and their potential role in diseases, other groups are focussing on biochemical or biophysical aspects of the connexin proteins e.g. channel structure, assembly, degradation and permeability. Shoji Maeda (University of Hyogo) showed the X-ray structure of the Cx26 protein at a 3.5 Å resolution. Bruce J. Nicholson (University of Texas) presented the differences in permeability of different connexin channels (Cx43, Cx32 and Cx26) and their selectivity for molecules (AMP, cAMP, GTP, cGMP). A particularly interesting study was presented by Stefan Wallner (LMU Munich, Germany) who showed data that microRNA molecules can be transported via gap junctions in HeLa cells. microRNAs have only been recently described and are known to regulate gene expression.

Our group's research (Imperial College London) is focussed on connexins in heart tissue. Decreased Cx43 expression occurs in ischemic heart disease and contributes to arrhythmogenesis. One section of the conference was entirely dedicated to connexins in the heart and was introduced by keynote speaker Andrew L. Wit (College of Physicians and Surgeons of Columbia University), who presented an overview of arrhythmogenesis and gap junction remodelling in heart disease from the last decade. Robert Gourdie (University of South Carolina, Charleston) presented data showing that a peptide binding to the C-terminus of Cx43 disrupts the association of the protein with ZO-1. This results in a decreased relocalisation of Cx43 to the lateral membrane of the cardiac myocytes which is a prominent feature in gap junction remodelling in heart disease. Giselle Rowlinson (Imperial College London) from our group presented data of Cx40 expression in the right ventricle of patients with congenital heart malformations. In the healthy ventricle only Cx43 is found, but in samples with double chambered right ventricle or tetralogy of Fallot, Cx40 is significantly up-regulated.

There is poor understanding of how changes in connexin expression and localisation of gap junctions occur. Connexin interacting proteins are thought to play a major role in regulating the connexin pathway, i.e. translation, transport to the membrane, gap junction assembly, gating mechanisms and degradation. Several talks described new interaction partners in many different cell types and a whole section was dedicated to this topic. Vandana Verma (University of Michigan) showed that small peptides binding to the C-terminus of the Cx43 protein inhibit the closure of gap junction channels and could therefore prevent the blocking of the action potential that occurs in arrhythmias. Investigation of molecules that reduce arrhythmogenic potential could have an impact on future therapy.

During the conference, two poster sessions were held in the hotel and enabled primarily younger researchers to show their data and discuss their findings. More then 50 posters were presented and the sessions were informative and lively despite the heat. Two members of our group presented data in the first of two poster sessions. I presented data that described caveolin-3 and caveolin-1 as Cx43



interacting proteins in the human heart. Since the mechanism of down-regulation of Cx43 in heart disease is poorly understood, all Cx43 interacting proteins could potentially play a role in these alterations. Thomas Desplantez (Imperial College London) presented two posters describing the electrophysiological characteristics of two cell lines established in our laboratory to investigate connexin co-expression and a characterisation of the connexin co-expression found in cell pairs of atrial myocytes using the dual voltage clamp method.

On the last night of the conference, a dinner banquet was organised in a restaurant close to the hotel. Afterwards many attendees ended singing karaoke in the Full Moon Saloon.

In conclusion, this meeting provided me with a great opportunity to present some of the data I have obtained during my PhD at an international meeting. I met many established researchers and learned about the research performed by many groups around the world. As a final year PhD student, attending this conference was also very valuable for my future career. I would like to thank the BSCB for awarding me with the Honor Fell Travel award which made it possible for me to attend the International Gap Junction Meeting.

Katharina Grikscheit
Imperial College London

XIII International Congress of Protistology

23–28 August, 2009. Armação de Búzios, Brazil.

The protists are a vastly diverse group of micro-organisms – encompassing hugely ecologically important species of photosynthetic algae and soil predators, as well as organisms responsible for devastating diseases of animals and plants. This diversity was particularly evident during the week of 23 to 28th of August (2009) when over 700 scientists gathered together for the XIII International Congress of Protistology.

This four-yearly meeting coincided with the XXV Annual Meeting of the Brazilian Society of Protozoology and the XXXVI Annual Meeting in Chagas' Disease, and discussed recent major advances in all things protozoan – from basic molecular and cell biology, through ecology and evolution to chemotherapy, vaccine development and vector control. The meeting was staged at the beautiful beach town of Armação de Búzios (just Búzios to the locals), 170km north of Rio de Janeiro, Brazil. Although the location was a great one, the weather conspired against leisure in favour of science and guaranteed that the attendees remained inside at the talks rather

than escaping to one of the 20 neighbouring white-sand beaches.

The meeting was opened by Walter Coli (Universidade de São Paulo, Brazil) who gave an in-depth global view of *Trypanosoma cruzi* research, from its discovery exactly 100 years ago to the most recent epidemiological results that celebrate no new reported case of Chagas' disease since 2005. Despite this good news, scientists were reminded to the need of effective treatment for the ~2.5 million people already infected. When *T. cruzi* has reached the heart of these infected humans, a progressively debilitating disease – cardiomyopathy – becomes certain, and no treatment is available other than heart transplantation. Until now; that is: Antonio Carlos Campos de Carvalho (Instituto Nacional de Cardiologia, Brazil) showed us that bone marrow stem cells can regenerate ventricular dilatations and improve cardiac functions in pre-clinical and clinical settings. He is currently coordinating a large, randomized clinical trial with 1500 patients in Brazil. If successful, this project would show the potential use of adult stem cells in cellular therapies, as well as representing a real avenue for treatment of Chagas' disease.

The daily schedule consisted of a general plenary early in the morning, followed by poster presentations until lunch time, and concurrent platforms of oral presentations and symposia in the afternoon. There was plenty to keep us busy throughout the entire programme of talks, yet most attendees found the energy to enjoy a couple (if not more) caipirinhas in one of the many seafront bars and restaurants.

Jan Tachezy (Charles University in Prague, Czech Replublic) gave the first plenary, discussing what might have been the mutual benefits for the eukaryotic host and its proteobacterial endosymbiont that led to the evolution of mitochondrion. He favoured the FeS cluster assembly pathway as the benefit which might have raised the original selection pressure for retaining an endosymbiont by the host. Thomas Cavalier-Smith (University of Oxford) discussed deep phylogeny of protozoa and the evolution of body plans, with special reference to the still uncertain position of the root of the eukaryotic evolutionary tree. While the plenary of Jean François Dubremetz (Université de Montpellier, France), in the last day, discussed how apicomplexan parasites (such as the causative agents of malaria and toxoplasmosis) invade the mammalian host cell, and how the structure of the latter is remodelled by this event. He highlighted the contributions of enzymes, especially kinases and pseudo-kinases, that are involved in changing the host cell genome expression.

The beginning of the afternoon was characterized by a difficult decision-making process – with 4 parallel programmes of symposia and oral presentations, it was common to see participants jumping from one room to the next in order to attend the most relevant talks in each platform. I had a particular interest in cell biology and eukaryotic evolution, so the symposium entitled "Free-living relatives of parasitic protists" was especially enjoyable. This comprised of talks on comparisons between genome sequences from diverse free-living eukaryotes, giving insights into the core features of the long-extinct last common eukaryotic ancestor (LCEA). Joel Dacks (University of Alberta, Canada) reconstructed the membrane-trafficking machinery present in the LCEA using large-scale multigene analysis and proposed that the ancestral eukaryote appears to have possessed a sophisticated cellular trafficking system. Scott Dawson (University of California Davis, USA) presented the newly sequenced genome of *Naegleria* (one of the few genome projects from free-living heterotrophic protists that can help uncover early evolutionary events) and extended the idea that the LCEA also possessed complex metabolic and signalling systems. While Andrew Jackson (Wellcome Trust Sanger Institute) presented the on-going genome sequencing project of *Bodo saltans*, the most closely related free-living protist to the human parasites of the genera *Trypanosoma* and *Leishmania*, and identified the genomic changes that accompanied the evolution of parasitism in this phylum.

Among the poster abstracts selected for oral presentation, highlights for me included Lillian Fritz-Laylin (University of California Berkeley, USA), Alex Paredez (University of California Berkeley, USA) and Helmut Plattner (University of Konstanz, Germany). When *Naegleria* (a predatory soil amoeba) metamorphose from an amoeba into a biflagellate (a change that takes <1.5h) actin synthesis ceases and the *de novo* assembly of an entire cytoplasmic microtubule cytoskeleton, basal body and axoneme is initiated. Using the newly sequenced *Naegleria* genome (see more above), phylogenetic profiling and microarray analysis, Lillian was able to identify 36 novel flagellar-associated genes and 38 potential basal body genes, which are currently under functional validation and characterization. At the level of cell signalling, Helmut presented some 'unorthodox' Ca^{2+}-release channels that are used by malaria parasites (Paramecium spp.) to exocytose secretory vesicles involved in host cell invasion. While Alex described the role of 3 divergent actin-related proteins found encoded in the *Giardia* genome. These proteins localize to the nucleus of the parasite and might be involved in chromatin remodelling. I also presented an oral presentation. This work (recently published in *PNAS*) described how specialized microtubules demarcate a membrane channel that traverses the protective boundaries of the extracellular parasite *Trypanosoma brucei* from attack by the human innate immune system. I also suggested, through a series of endocytic assays, that this channel is the major route of transport of nutrient macromolecules into the lumen of the flagellar pocket – a surface membrane domain in charge of all endocytosis and exocytosis in these parasites.

Parallel to the meeting, the organizers Marcia Attias (Universidade Federal do Rio de Janeiro, Brazil) and Renato DaMatta (Universidade Estadual do Norte Fluminense, Brazil) run the School of Advanced Studies, which placed 25 Brazilian graduates in close contact with some of the prominent speakers at the meeting. The School was focused on core seminars combined with smaller tutorials and discussion groups pre- and post-meeting, as well as during the evening in Búzios. This represented a great opportunity to those graduate students to discuss particular aspects of protozoan biology not covered by the meeting programme, as well as a chance to present their own specific research topics and ask particular questions regarding techniques which could aid their research. My impression was that the School was an extremely beneficial approach – both from the students and speakers' points of view – and I hope to see such initiatives becoming more frequent in international scientific meetings.

The final day of talks finished by midday, so attendees managed to squeeze in a last trip to the beach before catching the bus back to Rio de Janeiro airport. As if on cue, the weather dramatically improved and those lucky enough to be spending a few extra days either in Búzios or Rio could enjoy the beaches before returning home (in my case to a wet and grey UK). Overall the meeting was a fantastic opportunity to discuss how to make a greater impact on drug and vaccine research involving neglected parasitic diseases, as well as on general cellular biology. I am extremely grateful to BSCB for awarding me the Honor Fell Travel Award so that I could meet the travel costs to attend this highly informative and enjoyable meeting.

Catarina Gadelha
University of Cambridge.

Dicty 2009

23–28 August 2009. Estes Park, Colorado, USA

Researchers from all over the world gathered in the small town of Estes Park, Colorado, for the annual *Dictyostelium* conference. After meetings in Germany (2007) and Japan (2008) it was the turn of an American group to host this conference again, and the organisers Alan Kimmel and Tian Jin (NIH, Bethesda, USA) decided to convene at the foot of the Rocky Mountain Range.

We arrived in Denver, the mile high city, on Saturday, the 22nd of August, and could acclimatise to the high altitude for a night before taking the long bus trip to Estes Park the next morning. Estes Park itself is a popular summer resort at the Big Thompson River that houses the Stanley Hotel, which inspired Stephen King to the location for "The Shining". The town is also at an altitude of 7,522 feet, which was quite hard to get used to. But the amazing scenery surely made up for this.

The conference was held at the Rocky Mountain Park Holiday Inn hotel and began on Sunday evening with an informal reception where the 106 participants could familiarise themselves with each other and the location. Over the next four days, presentations were grouped into seven sessions.

Monday morning started with a general interest session on "genomics and whole-genome based analyses". Of most interest to me here was a talk by Rob Kay (MRC Laboratory of Molecular Biology, Cambridge) on "Sexual genetics and genome sequencing". Apart from identifying and sequencing of the mating type locus of *D. discoideum*, he reported resequencing of strain Ax4 that led to the discovery of thousands of errors in the full sequence available on the dictybase database, which is currently used as a reference by a huge number of researchers. Additionally, the group sequenced the widely used strain Ax2 and its parent strain DdB and found points of divergence between all strains, meaning that researchers have to be more cautious in using the sequence provided by dictybase without questioning its relevance to their particular strain.

Other topics of particular interest in the first session included Elizabeth Ostrowski's (Rice University, USA) "whole genome sequencing of natural isolates of *Dictyostelium discoideum*", who found polymorphisms between 12 different wild isolates of *D. discoideum* and closely related species in about 0.1% of the genome, and Anup Parikh's (Baylor College of Medicine, USA) "transcriptome conservation of the *D. discoideum* and *D. purpureum* developmental programs revealed through RNA sequencing", which introduced a new database called dictyexpress that provides mRNA sequences for both those strains. The session concluded with Yulia Bushmanova from the dictybase team introducing the improved usability of the newest version of the dictybase website interface.

Other speakers in the first session were Pauline Schaap (University of Dundee), Hideko Urushihara (University of Tsukuba, Japan) and Thomas Winckler (University of Jena, Germany).

The afternoon was dedicated to a session on actin-related processes. Speakers in this session were Chang Y Chung (Vanderbilt University, USA), Jianshe Yan (NIH, USA), Jelena Pribic (Hunter College, USA), Peter Thomason (Beatson Institute for Cancer Research, Glasgow), Rebecca Fernandez (Hunter College, USA), Annette Müller-Taubenberger (Ludwig Maximilians Unversity Munich, Germany) and Ben Rogers (Cardiff University).

The first day came to a successful conclusion in the first of two poster sessions.

Richard Gomer (Rice University, USA) began a session on signalling pathways and cAMP responses on the second day with his talk on a chalone signalling pathway. Talks by Jeff Hadwiger (Oklahoma State University, USA), Xin-Hua Liao (NIH, USA), Xuehua Xu (Georgetown University, USA) and Rick Firtel (University of California, San Diego, USA) followed, then Peter Devreotes (Johns Hopkins University, USA) presented "signalling events in chemotaxis". The session was concluded by Chris Janetopoulos (Vanderbilt University, USA).

The focus of the afternoon was on modelling cell motility. Edward Cox (Princeton University, USA) and Scott Gruver (Vanderbilt University, USA) were followed by Adrian Harwood (Cardiff University), who explained visualization of Dictyostelium chemotaxis by optical coherence tomography, which allows following cells moving on opaque surfaces or in a 3D environment. After talks by W F Loomis (University of California, San Diego, USA), Daniel Lusche (University of Iowa, USA) and Wouter-Jan Rappel (University of Michigan, USA), Peter Van Haastert (University of Groningen, Netherlands) detailed pseudopod formation during movement and chemotaxis using a computer algorithm to model cell movement. Finally, Deborah Wessels (University of Iowa, USA) brought up the possibility of a second chemotaxis system overlapping the cAMP system during cell aggregation. This topic was heavily discussed, before conference participants moved on to the second poster session.

On Wednesday morning, Ludwig Eichinger (University of Cologne, Germany), Salvatore Bozzarro (University of Turin, Italy), Adam Kuspa (Baylor College of Medicine, USA), Debbie Brock (Rice University, USA), Laurence Aubry (CEA Grenoble, France) and Steve Charette (Hôpital Laval, Canada) presented their findings on bacterial recognition and endocytic functions, before attendees parted to enjoy the long awaited excursions in the afternoon. There was a choice between white water rafting on the Poudre River and a hiking tour in the Rocky Mountain National Park, which were both well received. Unfortunately, I had recently injured my knee and had to stay behind. However, this allowed me extra time to improve and revising my talk planned for the next day.

In my opinion, Thursday morning saw most interesting session of the week, "Modeling of Human Disease-Related Pathways/ Developmental Recognition". My roommate Vanessa McMains (NIH, USA) reported that presenilin-signalling regulates growth and cell fate patterning in *Dictyostelium*, making it an adequate novel model

system for functional studies of the presenilin/gamma-secretase complex. Interestingly, she showed that, while *Dictyostelium* does not possess indigenous amyloid precursor protein (APP), the presenilin/gamma-secretase complex formed in the model system will process APP introduced externally. This is a promising result for the use of *Dictyostelium* to model processes occurring in Alzheimer's disease.

The second talk of the session was given by Robin Williams (Royal Holloway Unversity of London), my supervisor, on "PLA2 inhibition and lipid signalling in *Dictyostelium*: characterizing therapeutic targets in VPA treatment". After this, it was my turn to present – my first talk at an international conference! Despite being nervous, my talk on understanding short chain fatty acid uptake mechanisms in *Dictyostelium* was well received, and I got a lot of positive comments and suggestions in the following coffee break.

My talk was followed by Michael Myre (Harvard Medical School, USA), who investigated a huntingtin ortholog in *Dictyostelium* and its role in multicellular development. This topic was of particular interest to me, since my MSc project at King's College, London, involved a number of polyglutamine proteins found in *Dictyostelium* and investigating why these are tolerated so easily by *Dictyostelium*, whereas an increased size of the polyglutamine region of huntingtin leads to Huntington's disease.

Another interesting talk in this session was delivered by Gad Shaulsky (Baylor College of Medicine, USA), who reported on mechanisms of cheating and counter-cheating found in *D. discoideum*. These results were later reported in *Nature* later. Other speakers on Thursday morning included Christophe Anjard (University of California, San Diego, USA), Christopher West (University of Oklahoma, USA) and Chris Sugden (Dundee University).

The last session of the conference on Thursday afternoon was dedicated to "Vegetative Processes and Cellular Interactions" and included talks by Ralph Gräf (University of Potsdam, Germany), Douglas Robinson (Johns Hopkins University, USA), Harry MacWilliams (Ludwig Maximilians University Munich, Germany), Shigenori Hirose (Baylor College of Medicine, USA), Daniel Dickinson (Stanford University, USA), and Cynthia Damer (Central Michigan University, USA).

The conference was concluded with a banquet held outside the hotel and subsequent stargazing at the Estes Park Memorial Observatory, where we were joined by hobby astronomers from the local area who showed us a few constellations and willingly answered every question.

I am very grateful to the BSCB for supporting travel to the international *Dictyostelium* conference. Attending this conference enabled me to meet international leaders in the field and to get advice on my presentation and overall project. I returned to Royal Holloway with a greater appreciation of the importance of my research, and thus, greater motivation, as well as new ideas for future experiments.

Nicole Terbach
Centre for Biomedical Sciences
Royal Holloway University of London

FASEB Summer Research Conference on 'Mitosis: Spindle Assembly and Function'

30 August – 4 September 2009. Il Ciocco Resort, Lucca, Italy

Il Ciocco Resort is set on a natural 2000-hectare park in Serchio Valley, right in the middle of Tuscany, Italy. One hour away from Pisa, Il Ciocco take us into a countryside journey and set us in the perfect mood to listen to excellent talks.

This meeting was focused on mitosis and on how spindle formation is achieved. There were nine sessions in total. The first session was on centrosomes and spindle pole structure. Both second and third sessions were on kinetochores, about the mechanisms that are used to establish proper kinetochore attachments and also about kinetochore function and checkpoint signalling. The fourth session was on spindle assembly and organization that was followed by a fifth session about microtubule dynamics, motors and spindle function. There was a very interesting session, the sixth one, on Lifetime Achievement, in which three excellent researchers, Michele Bornens, Mitsuhiro Yanagida and Maurizio Gatti, summarized their lifetime research work. The seventh session was on mitotic progression and regulation of mitotic exit. The eighth session was about chromosome segregation and mitotic exit. Finally, the ninth session was on how mitosis can be used as a pharmacological target. In this five-day meeting there were so many outstanding talks and posters that I cannot mention all the interesting ones. So, I am just going to highlight some of the high points.

Erich Nigg (Biozentrum, University of Basel, Switzerland) was the keynote speaker of this amazing meeting and gave a talk on the cell cycle control of chromosome segregation. He has participated in a phosphoproteomics study of human mitotic spindles and discovered CHICA as an interactor of Kid, a chromokinesin that is required for the generation of polar ejection forces and chromosome congression.

CHICA localizes to the mitotic spindle and is both upregulated and phosphorylated during mitosis. Upon CHICA depletion, shorter mitotic spindles are formed, that failed to organize a proper metaphase plate, and Kid not longer localizes to the mitotic spindle. He also talked about recent work that he has done on the conversion of centrioles into basal bodies. He showed that overexpression of the centriolar protein CPAP leads to the formation of longer centrioles. The same phenotype was observed upon depletion of CP110, a distal-end-capping protein. By electron microscopy analysis he saw that the elongated structures that were formed upon CPAP overexpression or CP110 depletion were structurally different from normal primary cilia formed on RPE cells. He concluded that CPAP and CP110 play antagonistic roles in determining the extent of tubulin addition during centriole elongation. Finally, the keynote speaker also mentioned a work that he had done in the past regarding PICH (PLK1-interacting checkpoint helicase) protein. He had previously showed that this helicase co-localizes with PLK1 at kinetochores and inner centromeres and that it also decorates threads that form during metaphase. Upon depletion of PICH, there is a selective loss of Mad2 from kinetochores and a complete abrogation of the spindle checkpoint that ends up resulting in a massive chromosome missegreagtion. He has now realized that the three different batches of siRNAs that they were initially used to deplete PICH all had Mad2 as an off-target. He is now performing new experiments to understand whether PICH really plays a role in spindle checkpoint regulation.

David Glover (University of Cambridge, UK) gave a very interesting talk about the regulation of SAK/PLK4 in *Drosophila*. He started by showing that SAK/PLK4 is required for centriole formation, both canonical and *de novo*, being a master regulator of the process. He then showed recent work done on asterless, a centriolar protein that he showed to be required to load SAK/PLK4 for centriole duplication. SAK/PLK4 interacts *in vivo* and *in vitro* through the N-terminus of asterless and the cryptic polo box of SAK/PLK4. Similar to overexpression of SAK/PLK4, overexpression of asterless induces centrosome amplification in embryos and their *de novo* formation in unfertilized eggs. A SAK/PLK4-binding deficient asterless mutant localizes to centrosomes but fails to recruit PLK4 and with that suppresses centriole duplication.

Christine Suetterlin (UC-Irvine, CA, UAS) spoke about the mechanism behind centrosome overduplication that is observed upon *Chlamydia* infection. *Chlamydia trachomatis* causes intracellular infection and is involved in cervical cancer. It was already known that *Chlamydia* infected cells have supernumerary centrosomes and that it uses the canonical centrosome duplication pathway for that amplification. She showed that endogenous HsSAS-6, a centriolar protein required for centriole formation, is cleaved during *Chlamydia* infection. This cleavage takes out HsSAS-6 Ken box and expression of the cleaved form of HsSAS-6 induces centrosome amplification. This amplification is PLK4-independent because she showed that after PLK4 overexpression HsSAS-6 is not cleaved. She is now trying to identify the protease that cleaves HsSAS-6 upon *Chlamydia* infection. Interestingly, HPV infection also causes centrosome amplification but it is not known if it is through the same mechanism.

Zita Carvalho-Santos (Instituto Gulbenkian de Ciencia, Oeiras, Portugal) gave a very interesting talk on how comparative genomics and experimental analysis can be used to understand the evolution of centriole/basal body assembly machinery ability to build the same structure in different contexts. She identified a core of three components (SAS-6, SAS-4/CPAP and Bld10/Cep135), which was called UNIMOD, that correlates with the occurrence of centrioles/basal bodies and that can be used to determine the presence of this structure in other organisms. Surprisingly she saw that other centriolar components such as SAK/PLK4, SPD2/Cep192 and CP110 are not part of the UNIMOD and emerged in a taxon-specific manner. Interestingly, she showed that protein divergence and duplication leads to taxon-specific novelties. Divergence between

SAK/PLK4 family members leads to loss of cross-species complementation. *Drosophila* Bld10/Cep135 is one example of a protein that has two ancestral functions in centriole and axoneme biogenesis, that subfunctionalized in humans in two duplicates. In fact, she showed that *Drosophila* Bld10/Cep135 mutant males contain shorter centrioles and their axonemes lack the central pair. *Drosophila* Bld10/Cep135 is then required for male fertility, which probably reflects a tissue-specific role in axoneme biogenesis.

Robin Allshire (University of Edinburgh, UK) discussed CENP-A chromatin assembly and kinetochore formation. CENP-A is a highly conserved histone H3 variant that is found at centromeres, specifying sites of kinetochore assembly. It is known that there is an epigenetic determinant for CENP-A localization. He wanted to understand what marks centromeric DNA and makes it a prime site for CENP-A assembly. Using fission yeast minichromosomes plasmids he showed that Clr4-dependent centromeric heterochromatin influences the establishment of CENP-A within the central domain of the kinetochore. He also showed that thethering Clr4 via DNA-binding sites at euchromatic loci induces heterochromatin assembly. Hence, synthetic heterochromatin completely substitutes for outer repeats on plasmid-based minichomosomes, promoting de novo CENP-A assembly. This is very interesting as it shows that H3K9 methylation-dependent heterochromatin is alone sufficient to form functional centromeres.

Kevin Hardwick (University of Edinburgh, UK) gave a talk on how to assemble and disassemble anaphase inhibitors. The spindle checkpoint is a surveillance system acting in mitosis to delay anaphase onset until all chromosomes are properly attached to the mitotic spindle. It is known that when the checkpoint is active, the Mad2 and Mad3 proteins directly bind and inhibit Cdc20 leading to APC/C inhibition. He wanted to know if the activity of mitotic kinases need to be reversed by protein phosphatases before anaphase onset can occur. He used fission yeast to firstly show that Aurora kinase activity is directly required to maintain spindle checkpoint arrest, even in the presence of many unattached kinetochores. Upon Aurora inhibition the checkpoint complexes are disassembled and cyclin B is rapidly degraded. He further showed that the checkpoint signalling and cyclin B degradation require the kinetochore-localized isoform of protein phosphatase 1. He proposed that PP1-mediated dephosphorylation of checkpoint components forms a novel spindle checkpoint silencing mechanism.

In the end, this FASEB meeting on "Mitosis: Spindle Assembly and Function" proved to be very enjoyable and interesting, where it was possible to hear excellent scientific work, have the opportunity to present my work and meet many interesting people. My thanks go to the BSCB for the Honor Fell Travel Award which went towards the cost of attending this meeting.

Ana Rodrigues-Martins
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16th International Society of Developmental Biology Congress 2009

6–10 September 2009. Edinburgh, Scotland, UK

A beautifully picturesque and sunny Edinburgh welcomed over 1000 delegates to the 16th Congress of the ISDB with the aim of bringing together some of the key figures in all areas of developmental biology for this flagship meeting.

The conference was held at the state-of-the-art Edinburgh International Conference Centre, purpose built for events of this nature and scale. A short walk from both Edinburgh stations, it was a fantastic choice for the meeting by the conference committee. As a first year postgraduate, the sheer size of the meeting made it a unique experience with session of at least 3 lecture theatres running concurrently for the entire duration of the conference, there was plenty going on to keep me entertained.

The conference began with 2 fantastic plenary lectures. In the first, the 1995 Nobel prize winner Prof Eric Wieschaus, Princeton (Patterning transcription and cell shape change in the *Drosophila* embryo), described his seminal work looking at the cell fate decisions made in the very earliest stages of *Drosophila* development. This was followed by an interesting plant development lecture by Prof Caroline Dean, Norwich (Vernalization - cold-mediated epigenetic regulation of a developmental switch).

I then attended the symposium on 'Non-coding RNA in development' as I have an interest in ncRNAs due to their pivotal role in imprinting at several gene loci. There were workshops on a wide-ranging set of topics for the morning and afternoon session covering diverse aspects of developmental biology, with growth control, morphogenesis and embryonic induction the key themes explored on the first day alongside the ncRNA and Epigenetics symposia that I chose to attend. I found the Epigenetics lectures particularly interesting and insightful and the lecture by Prof Wendy Bickmore exploring the relationship between epigenetic histone modification and hox genes, linked nicely the themes of epigenetics and development.

The first day drew to a close with another fantastic plenary lecture by Dr Liz Robertson, Oxford, UK, who received the 2009 Waddington Medal for outstanding research performance and services to the subject community. Her biographical talk described her life and work, and took us right from her childhood- barefoot in Africa, where her father was working as a botanist, to her undergrad studies at Oxford, postgrad at Cambridge and eventual Professorships at Harvard and Columbia Universities. Her research was decisive in shaping the tools used by molecular biologists today, particularly with regard to the engineering of knockout mouse models. It was an inspirational lecture, particularly for young researchers like myself who are just embarking on our research careers.

The second day began with a dull headache, possibly due to sampling the Edinburgh nightlife the night before, but fortunately the early morning lectures by Dr. Janet Rossant, Toronto and Professor Margaret Fuller, Stanford, were more than interesting enough to maintain the attention of a few flagging individuals. Dr Rossant's leture on the role of the Hippo signaling pathway was very interesting as it forms the theory for the first signal of epiblast cell differentiation in early mouse development.

Day 2 continued with symposia on the cell fate, organogenesis and asymmetry comprising the main themes. However, it was the Graduate Student Symposium that particularly caught my eye. Few meetings of this size present such a comprehensive forum for young delegates to present their research and findings. This 2 hour session gave 6 postgraduates a chance to talk to peers and lab heads about their work, and was a real plus point for me regarding the conference.

The last full day of lectures took a far more translational approach to developmental biology research, with lectures on stem cells in medicine, disease factors and pluripotency the topics of the day.

The factors that contributed to the success of the meeting were the exceptional organization; the sheer scale of the meeting meant that anything less than military style organization would have resulted in disaster. My initial fears that that I would feel swamped by the information being sent my way was allayed by the small parallel symposia settings, and far from feeling alienated by the scale the meeting gave me great opportunities to meet other people interested in my subject area and research goals!

6 poster-viewing sessions allowed me time to mingle with the other delegates and I am now entering into collaboration with University of Bath due to initial contact made at the ISDB 2009 during a poster session. Having been aware of the group's work through their recent publications, the conference gave me the ideal opportunity to speak to the primary author of the poster and discuss my ideas into imprinting of the gene Grb10. I am now using 2 knockout lines from the group to assay several potential mechanisms, which I hope will lead to a publication, and will certainly make up a chapter of my thesis.

The meeting drew to a close on Thursday with a fascinating lecture by Dr Masatoshi Takeichi, Kobe Japan, on the role of adherin junctions, which are contact proteins between cells important in cell migration during morphogenesis. I also had time to explore the beautiful city of Edinburgh whilst it was still light, and to eflect on a successful and intriguing meeting.

The ISDB conference happens every 4 years with the next meeting scheduled for 2013. It is a 'must attend' for those for whom developmental biology plays any role in their research as the broadness of scope really helps give you perspective to the context of your project. I would like to thank the BSCB for their generous award of the Honor Fell Travel grant, which allowed me to attend this very useful meeting.

Adam Prickett
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International Symposia on Signalling at the Blood-Brain and Retinal Barriers

9–11 September, 2009. University College London

The 12th instalment of the International Symposia on Signalling at the Blood-Brain and Retinal Barriers was a great success. With a new record attendance of 148 delegates from Europe, the Americas, Asia and Australia, the status of this meeting series has undoubtedly grown further.

Thirty talks were presented in scientific sessions concentrating on Pathology and Disease, Homeostasis, Cell Passage, Cell Biology, Transport, and Junctions and Permeability. An even mix of invited speakers with international reputation and others chosen from submitted abstracts guaranteed a versatile programme of excellent scientific value. Another 67 abstracts were presented as posters, all of which were of high and competitive standard, and subject to lively debates during the three poster sessions.

The feedback on the quality of the conference was fantastic; some comparing this conference with its US counterpart, the Gordon Conference on BBB. In an end-of-conference survey 89% of delegates declared to have attended an excellent or at least very good conference. In particular the keynote lecture given by Prof Elisabetta Dejana (Milan, Italy) on BBB development was particularly praised. Similarly, the sessions on Disease, Cell Biology, Cell Passage and Junctions were judged by at least 80% of delegates as either very good or even excellent.

In conclusion this has been an exciting meeting on the latest developments in research of blood-neural barriers. Undoubtedly, it has provided continuous insight into the pathology and future treatment of neuro-vascular diseases such as MS, epilepsy, stroke and neural inflammation.

Maria Balda
University College London

Endocytic machineries in control of cell signalling and tissue morphogenesis

3–8 October 2009, Panorama Hotel, Chania, Greece

Set in the beautiful Cretan town of Chania, the Hotel Panorama was an ideal venue for the first biennial "Endocytic machineries in control of cell signalling and tissue morphogenesis" meeting. This meeting brought together over 150 scientists researching endocytosis, vesicle trafficking and cell signaling.

Part of the EMBO conference series on "Membrane dynamics in Endocytosis" this conference was dedicated predominantly towards examining the function of multi-protein complexes in endocytic vesicle trafficking, cell signaling and tissue development. The conference was organized by Harald Stenmark (Centre for Cancer Biomedicine, Norway) and Gillian Griffiths (Institute for Medical Research, UK) both of whom gave wonderful presentations illustrating exciting unpublished observations from their own labs. The meeting was attended by representatives from all research levels each with common interests in various aspects of intracellular

trafficking. The conference commenced on Saturday evening with registration followed by dinner. This was the perfect opportunity to meet with fellow conference attendees and begin what was to be an intellectually packed few days.

On the Sunday morning the first session was opened with a talk delivered by Tomas Kirchhausen (Harvard Medical School, USA) who gave a highly insightful talk about molecular mechanisms involved in uncoating of clathrin coated vesicles. This was followed by a talk by Frances Brodsky (University of California, San-Francisco, USA) who discussed her work on the role of the CHC22 Clathrin Heavy-Chain isoform in trafficking of the GLUT4 glucose transporter. Her work shows the requirement for CHC22 isoform in the formation of insulin-responsive GLUT4 compartments in human muscle and adipocytes, and the potential implications for type 2 diabetes.

Christophe Lamaze (Institute Curie, France) presented work on the role of caveolae in cell mechanosensing. Their recent work examining plasma membrane tension in mouse endothelial cells suggest a role for caveolin-1 in regulating membrane tension. Using plasma membrane nano-tubes, uniaxial stretching, and hypo- and hyper-osmotic shock, this group show increased freely diffusing caveolins in the plasma membrane of cells undergoing mechanical stress. This innovative work suggests disassembly / reassembly of plasma membrane caveolae as a primary response to plasma membrane stress. The subject of caveolae was raised again by Robert Parton (The University of Queensland, Australia) whose presentation focused on caveolae formation. His work showed Cavin1 (PTRF) colocalises with caveolae and that expression of caveolin-1 in caveolin-null cells led to re-localisation of cavin to the plasma membrane in a manner dependant upon cholesterol. Work utilizing the Zebrafish notochord showed knockdown of cavin1 inhibited formation of caveolae on the plasma membrane. This work suggests a role for cavin1 in the biogenesis of caveolae. Christopher Burd (University of Pennsylvania School of Medicine, USA) presented an excellent talk on the trafficking of the Fet3/Ftr1 yeast reductive iron transporter. Interestingly localisation and recycling pathway of the Fet3/Ftr1 transport differs depending whether iron is bound. His work showed correct sorting to multivesicular bodies requires ubiquitination by the Rsp5 ubiquitin ligase whilst sorting via recycling pathways is mediated by the retromer complex and the sortin-nexin Snx3.

Mark McNiven (Center for Research into Digestive Diseases, Mayo Clinic USA) gave a wonderful talk on the degradation of the epidermal growth factor (EGF) receptor via regulation of late endosomal budding mediated by the Dynamin2-CIN85 complex. This work shows that Dyn2 and CIN-85 serve as an adaptor protein during ubiquitin mediated down regulation of EGF receptors. Inhibition of this complex led to delayed EGF receptor degradation and elongated Rab7 positive endosomes. This suggests a function for dynamin 2 in trafficking of the EGF receptor at later stages of the endocytic pathway.

An interesting talk on the function of Arf6 (ADP-ribosylation factor 6) in the endocytic recycling pathway was given by Philippe Chavrier. Using TIR-FM (total internal reflection fluorescent microscopy), dominant-negative and dominant-active Arf6 mutants (Arf6 T44N and Arf6 Q67L respectively) the group found only active GTP bound Arf6 mutants co-localised with clathrin coated pits (CCPs) and using FRAP found Arf6 recruitment is highly dynamic and dependant upon activation state. Further studies show AP2 is required to localize Arf6 to CCPs. Interestingly however siRNA silencing of Arf6 inhibited recycling of some cargo proteins but not others. Knock-down of Arf6 decreased trafficking of the transferrin receptor but had no effect on EGF receptor recycling. This group suggested Arf6 plays a role in selective trafficking of cargo from the perinuclear recycling compartment back to the plasma membrane.

Too many speakers gave exciting and interesting presentations to be described in detail, but others that stood out include Marino Zerial (Max Planck Institute, Germany), Lukas Pelkmans (Institute of Molecular Systems Biology, Germany), Sylvie Urbé (The University of Liverpool, UK), Pier Paolo Di Fiore (The University of Milan, Italy), and Enrique Rodriguez-Boulan (Weill Cornell Medical College, USA).

The final speaker of session seven was the keynote lecture presented by Pietro De Camilli (Yale School of Medicine, USA). His exceptional presentation highlighted his work on regulation of clathrin mediated endocytosis and characterization of key molecules involved in this process, specifically within neurons. Session 8 was devoted to presentations from selected abstracts. This session gave a wonderful opportunity for post-graduate students and post-doctorial researchers to showcase their data to leaders of their field. This provided an excellent opportunity for feedback and advice. All sessions were highly interactive with many questions from the audience, some snowballing into highly interesting debates. To further audience participation and increase the diversity of questions, bar vouchers were offered as an incentive for questions from post-doctorial researchers and students. At the end of the first three days poster-sessions were organized. All posters were of an extremely high quality and sessions were highly productive and relaxed. My personal experience was that this meeting was an excellent opportunity to discuss both my own work and see what other people in related fields are doing. It was also good to discuss methods with people with far more experience than myself. I am sure that many project collaborations were forged both during poster sessions and the bar once poster presentations were over.

The final session was wrapped up with a traditional Greek meal, drinks and a disco. Poster prizes were awarded and Harald Stenmark and Gillian Griffiths closed the meeting. This final evening really was a wonderful way to end the last session giving everybody an opportunity to relax and enjoy the beautiful Aegean weather. The disco was a great hit, shortly after music began (even before dessert was served) many people were dancing and having a fun time. The

final day was a morning trip to Chania old town where people could spend time investigating the local area.

The "Endocytic machineries in control of cell signalling and tissue morphogenesis" conference was a complete success; its focused nature meant that all participants had common research which led active discussions during both session and social time. It was the first of its kind and would not be able to take place without its sponsors The Research Council of Norway, Cell Press, The Journal of Cell Biology, The European Molecular Biology Organisation and the Federation of the Societies of Biochemistry and Molecular Biology. I would like to thank both Harald and Gillian for organizing such a wonderful event. I also would like to thank both the BSCB for my receipt of the Honor Fell Travel Award and the BBSRC for funding my attendance at this conference. I am already looking forward to the next meeting, scheduled for 2011, and encourage anybody who was not able to get to Chania to attend the next meeting.

Sarah Fletcher
University of Birmingham, UK

The Mediterranean location Crete, with sunny days and temperatures >25°C, attracted an impressive line up of speakers to a very stimulating meeting with animated discussions. The meeting was intense, with talks starting at 9 am, a three hours lunch break, and an afternoon session lasting until 7 pm. After dinner, the very well visited poster session often went on until midnight.

The meeting was chaired by Harald Stenmark (The Norwegian Radium Hospital, Norway) and vice-chaired by Gillian Griffiths (CIMR, Cambridge, UK), who additionally gave a talk on polarity of endocytotic organelles in lymphocytes on the last day of the meeting. The meeting kicked off with Tom Kirchhausen (Boston, US), reporting on different aspects of clathrin-dependent endocytosis. Biochemical data was presented showing that the successive build-up of PIP2 levels in nascent clathrin-coated vesicles (CCVs) leads to recruitment of the chaperone Hsc70, which, upon reaching a certain threshold, initiates the dissociation of clathrin from the vesicle. This mechanism could explain why CCVs are uncoated only upon scission from the plasma membrane. As always in the clathrin field, this led to a vibrant discussion between different opinions. Frances Brodsky (UCSF, US) reported on an interesting finding that a functional clathrin heavy chain isoform (CHC22) expressed in human muscle and adipocytes is not expressed in mice. She showed that CHC22 is an important determinant in GLUT4 trafficking as transgenic mice expressing human CHC22 display abnormal glucose homeostasis with diabetic like phenotypes. These data are of major importance to the field, as most work to date on GLUT4 trafficking and glucose metabolism has been carried out in rodents. David Owen (CIMR, Cambridge, UK), a structural biologist among cell biologists, gave an interesting talk on how SNAREs (Vamp7 and Vti1a) are selected for incorporation into CCVs. He reported that unstructured regions in the N-terminus of SNAREs are recognised by specific adaptor proteins.

The meeting had several talks on exosomes (M. Simons, Gottingen, Germany; W. Stoorvogel, Netherlands and J. Gruenberg, Geneva, Switzerland) and the mechanisms of their generation from multivesicular bodies (MVBs), an exciting field that still appears in its early days. There were several talks on the structure and function of the ESCRT complex and endosomal sorting of ubiquitinated cargo. Scott Emr (Ithaca, US) presented a "conveyor belt model" for the inward budding and generation of intralumenal vesicles by ESCRT III. Ubiquitin, besides being a tag to target proteins for lysosomal degradation, has been shown to function in various other cellular context. V. Lobert (from the Stenmark Lab) identified a role for ubiquitination of integrins in fibroblast migration and S.Urbe (Liverpool University, UK) showed data on ubiquitin dynamics and signalling of growth factors, and thereby showed various new aspects on the functional dynamics of this important reversible modification. M. Miaczynska (Warsaw, Poland) reported on the function of endosomes as signalling platforms, a new and emerging field that highlights the dual function of endosomal proteins both in the endocytic trafficking and their role in nuclear signalling.

Two talks focused on the use of toxins as tools to investigate principal cell biological questions and to investigate toxin uptake into the cell as well as retrograde trafficking pathways within the cell (K. Sandvig, Institute for Cancer Research, Norway; L. Johannes, Institute Curie, France) and it is clear that these toxins use multiple endocytic pathways pathways to gain access to the inside of cells.

Several labs reported on the strength of high throughput systems biology approaches to tackle cell biological problems and the power of software-assisted computation to be able to make sense of the immense amount of data obtained in such screens (Marino Zerial, Dresden, Germany and T. Van den Hoorn, Amsterdam, The Netherlands). One talk of general interest in this respect was Lukas Pelkmans' (ETH Zurich, Switzerland) quantitative approach to unravel the contribution of cell-to-cell variability and cellular environment to cellular activities such as endocytosis. He showed that positional information of cells grown in culture can be used to predict (by Bayesian network learning) the efficiency with which different ligands (e.g. transferrin, SV40, cholera toxin) are internalized. He thereby emphasized the importance of taking into consideration the population-determined heterogeneity when planning an experiment. He then presented unpublished data showing that phosphorylation of focal adhesion kinase (FAK) is an important indicator of cell density, leading to a downstream signalling cascade that regulates the plasma membrane levels of GM1 (the receptor for SV40 and cholera toxin), thus ultimately determining the uptake and trafficking itinerary of SV40 and cholera toxin. J. Mercer from the Helenius lab (ETH Zurich, Switzerland) presented data on a high throughput siRNA screen against factors required for productive vaccinia virus infection. Several "drugable" cellular factors were identified, among which numerous genes involved in proteasome function and EGFR signaling. V. Marjomaki (University of Jyvaskyla, Finland) showed that Echovirus (a picornavirus) and its cellular receptor 21 integrin enter the cell via macropinosomes and traffic via neutral pH compartments eventually altering the structure of MVB.

I was selected to give a talk on the basis of my submitted abstract and rather dauntingly, I was scheduled to give my talk after the

keynote speaker Pietro De Camili (Yale University, US), who gave an impressive tour de force on his work on the molecular anatomy of early endocytic intermediates. He also presented recent data from dynamin I and dynamin II double knockout mice. Primary cells isolated from these animals displayed more clathrin coated pits (CCPs), which often appeared to be tubulated and showed a marked increase of components of the actin machinery that associates with CCPs during internalisation.

My talk was entitled "SDPR induces membrane curvature and functions in the formation of caveolae". This was the third talk directly addressing the function of caveolae. In my talk I focused on a family of caveolar proteins termed cavins. Our data demonstrate a functional interaction of two of these newly identified proteins in caveolae biogenesis, and the potential function of caveoale in shiga toxin uptake. This later effect led to an interesting discussion over lunch with the toxin lab heads. R. Parton (Brisbane, Australia) had earlier in the meeting shown data on one of the other cavin molecules during his talk earlier in the week. He had further illustrated that expression of caveolin-1 in a bacterial system appears to be sufficient to generate structures that morphologically resemble caveolae. Cristophe Lamaze (Institute Curie, France) alluded to caveolae function in machanosensing, supporting a role for caveolae in maintaining membrane tension. One speaker (H. Riezman, University of Geneva, Switzerland) addressed the significance of lipids in membrane function directly using yeast genetics and metabolic engineering and demonstrated the importance of functional interactions between sphingolipids and sterols.

The meeting ended with a gala dinner and a very well visited dance floor and the following morning the organizers had arranged a visit to nearby highlights, a trip missed by some possible due to the previous nights escapades.

I want to thank the organizers for an excellent meeting, co-participant Alex Ludwig for comments on the report and BSCB for supporting my trip to the meeting.

Carsten Gram Hansen
MRC-Laboratory of Molecular Biology
Trinity Hall, Unversity of Cambridge

Society for Neuroscience Annual Meeting

17–21 October 2009. Chicago, IL.

The annual meeting of the Society for Neuroscience is probably the best attended and widest ranging meeting on the neuroscience calendar. This year more than 30,000 people descended on Chicago to enjoy all that the meeting had to offer. Thanks to an Honor Fell Travel award from the BSCB I was able to join them.

The meeting began on Saturday morning on the light-hearted note with presentations by internationally renowned magicians Apollo Robbins and Eric Mead. Their impressive display of forcing a member of the audience to selectively forget a particular item in a list, and pick-pocketing another scientist from the audience was followed by an interesting discussion and question session on the neuroscience of memory and attention.

Later the same day there was a Special Lecture by Liqun Luo (Stanford University, USA), who discussed techniques developed for both Drosophila and mice that allow the visualisation and manipulation of specific neurons and neural circuits. Professor Luo described his methods that can label small numbers of neurons, and allows the study of the connectivity and functions of particular neuronal types.

The theme of neuronal circuit manipulation and formation was very strong at the meeting, and closely aligned with my own research interests. A symposium on Saturday afternoon, to showcase various new technologies for probing neuronal circuits with light, was extremely well attended, with many people having to stand at the back or sit on the floor around the conference room. Among the speakers was Mark Schnitzer (Stanford University, USA) who described some very interesting methods his lab had developed for imaging the activity of neuronal circuits in behaving animals. Maiken Nedergaard (University of Rochester, USA) went on to described her work on the role of astrocytes (glial cells found in the brain) in neuronal network activity, these cells are often overlooked in the study of the brain and this talk gave an interesting insight into the roles of these cells in neuronal circuit activity, as well as showcasing the methods that had been developed to study the activity of these cells in the nervous system.

The Sunday program was again packed with a variety of posters and symposia. The highlight for me though, was the Fred Kavli Distinguished International Scientist lecture given by Daniel Wolpert (University of Cambridge). Professor Wolpert made the case that the study of human motor control is one of the most important aspects of neuroscience, because ultimately movement is the main output from the brain. Although the talk contained aspects of human psychology and even Bayesian statistics, and so was somewhat removed from my own main interests, I found the seminar very engaging. This highlights one of the best aspects of the Neuroscience meeting – being able to explore different subjects that you would not normally be exposed to.

Monday morning began with looking at some of the hundreds of posters that were on display at any one time. This included one from S.W. Oh from the Allen Institute for Brain Science (USA), which described an ambitious project to begin mapping the connections of different neuronal types in the mouse brain. Using a combination of viral vectors activated by Cre-recombinase, transgenic mice and fluorescent proteins it was possible to label individual neuron types and trace their connections. If the success of the Institute's Mouse Brain Atlas is anything to go by then this project is likely to give a number of

important insights into the connectivity patterns of neurons. In the same session Sarah Rogan (University of North Carolina, Chapel Hill, USA) described her laboratory's method for controlling the activity of neural circuits by expression of DREADD receptors in particular neuron types. This receptor has no endogenous ligand in mice, but by driving its expression in specific neurons and giving an intraperitoneal injection of its ligand the activity of these neurons could be selectively altered.

After the poster session I had the chance to experience a new format for presentations that had been introduced this year. Called nanosymposia, these were designed to be a group of around 10 abstracts that were linked by topic (or even from the same laboratory), with 10 minutes of presentation and 5 minutes of questions. They were specifically aimed to give graduate students and postdoctoral fellows a chance to present their work in a smaller setting than in a full symposium. I attended the axon regeneration nanosymposium. Among the talks was one from Kevin Park of Zhigang He's laboratory (Harvard Medical School, USA), who described how they could manipulate the mTor signalling pathway with adeno-associated viruses to promote regeneration of damaged retinal ganglion cells. The format seemed to work very well and I'm sure it will be a feature of future meetings.

The scientific sessions each day were concluded by the Presidential Special Lecture. These featured some of the most renowned neuroscientists from around the world and usually made a very interesting conclusion to each day. By far the best attended of these, and probably the most widely attended session of the whole conference was the Presidential Special Lecture by Eric Kandel (Columbia University, USA). Professor Kandel's long and distinguished career has focussed on studying the mechanisms underlying memory formation. The session began with a description of Professor Kandel's career, given by Tom Carew (University of California, Irvine, USA), the current president of SFN and a former member of the Kandel laboratory. Professor Kandel then described his work in both the sea slug Aplysia and in mice that has really shaped our current understanding of how memories are encoded and stored in the brain. The talk was inspiring and for me one of the main highlights of the conference.

NEUROSCIENCE 2009

The final day of the conference was when I was due to present my own poster. Being scheduled on the last day I had been concerned the attendance would be much reduced as many delegates took the opportunity to catch an early flight home, or to explore Chicago one last time. However, the meeting was as busy as it had been all week. My session started at 8 am and I received a lot of interest in my work, and spoke to a continual stream of people until my session was over. I received a number of interesting comments and suggestions that were extremely useful and helpful. Many of the comments I received were particularly helpful in that they came from people outside of my normal field and gave a different perspective on the work, reflecting the wide variety of interests and specialities that are represented at SFN. After the poster session it was time for me to go to the airport and catch my flight home. My poster session turned out to be the perfect end to what had been an interesting and exciting meeting, and I thank the BSCB for their support, which made my attendance possible.

Andrew Murray
School of Medical Sciences,
University of Aberdeen

With the attendance topping 30,500, this conference was international neuroscience on a grand scale. Thanks to a Honor Fell travel award form the BSCB I was able to attend this year's SFN conference. With its symposia and poster sessions on topics ranging from diversity of synaptic plasticity to preference and aversion learning, SFN Chicago 2009 was always going to be an interesting and enjoyable experience.

The start of the conference is always a pleasant experience with an afternoon session to ease you into the frenetic pace that is set for the rest of the conference. There were a number of interesting poster session throughout the afternoon. Sessions included adult neurogenesis and stems cells, CNS and PNS regeneration and inflammatory mechanisms and cytokines in demyelinating disorders. A particularly interesting poster from T. Suter (University of Zurich) showed that NG2 was abundant around infiltrating leukocytes in spinal cords of EAE mice suggesting a possible detrimental role for NG2. They used NG2 knockout mice to show improved recovery in relapsing remitting phase suggesting the increase of NG2 by OPCs and macrophages following injury may augment axonal damage and demyelination. Saturday afternoon also included a poster session on oligodendrocyte differentiation, which included a number of interesting posters including one from Vittorio Gallo (Children's Hospital, Washington DC). Vittorio's group examined the molecular mechanisms of why, following perinatal hypoxia, although oligodendrocyte progenitors proliferate they fail to mature. They identified decreased expression of p27kip1 results in decreased maturation and increased BrdU incorporation, thus this is a key regulator responsible for limiting oligodendrocyte maturation following perinatal white matter injury.

Sunday morning for me began with a minisymposium session on axonal transport defects in neurodegenerative disease. This was a diverse session with talks encompassing the abnormal axonal transport mechanisms in Parkinson's disease, Alzheimer's disease and Amyotrophic lateral sclerosis. As always the poster session of the

morning was busy and in typical fashion many of the posters I was interested in were at opposite ends of the centre. Philip Rhodes (University of Mississippi) presented a particularly interesting poster whereby they showed that dexamethasone, typically used to treated pulmonary dysplasia in premature infants, has a detrimental effect, inducing caspase-3 activity and TUNEL positive cells in a rodent model. There was also a poster from Stephen Goldman's group (University Rochester). I had previously been lucky enough to attend a lecture by Stephen and their work on human glial progenitors to generate a chimeric mouse is very elegant, whereby over time the human glia A2B5+ cells expand outward and over the course of 5 months the human GRPs out compete co-transplanted mouse GRPs in the Rag$^{-/-}$ Shiverer transgenic mice. I had just enough time to grab some lunch before I presented my poster in the afternoon session on the neuroprotective effect of oligodendrocyte precursor cells in perinatal white matter injury. I have previously found that staying by your poster is always beneficial and so it proved this time with a number of delegates attending my poster providing me with a good opportunity to gain both ideas for future experiments and potential collaborations.

The third day of the conference was packed with a variety of interesting and entertaining symposia and poster sessions. There were a number of interesting posters by the same group examining the beneficial effects of berry fruits – in particular red raspberries, which had a beneficial effect on reducing age related decline in motor and cognitive behaviour. The second was the use of walnuts to reduce the inflammatory response by microglia following exposure to LPS and thus may be a potential therapy against brain inflammatory diseases. Also taking place on Monday was a symposium, which was of particular interest to myself on the role of oligodendrocyte precursors in neurological diseases, presented by a number of the eminent scientists of glial cell biology including Patrizia Casaccia-Bonnefil, David Pleasure, Vittorio Gallo. Vittorio Gallo spoke about the response of the oligodendrocyte precursors both in development and demyelinating diseases and how the progenitor pool is affected by loss and damage. Vittorio highlighted the importance of EGFR signalling in coordinating the response of the progenitors and how the repopulation following injury occurs from progenitors in the SVZ

Tuesday was punctuated by various nanosymposia and poster sessions. As always time is never on your side at these large conferences and whilst many of the posters I passed appeared interesting I only had a short time frame to make sure I visited those that I had identified on the itinerary planner prior to going to Chicago. Of particular interest were three posters. The first was from M Dragunow's group (University of Auckland, New Zealand) who illustrated for the first time that Valproic acid, used for treatment of mood disorders and epilepsy, decreased the expression of microglial markers and reduced their phagocytic ability which may result in reduced ability of healthy brain to respond to inflammatory diseases such as Multiple Sclerosis. The second was from S Juliano (Uniformed Services University, Bethesda) who made use of injured or non-injured paradigms in rat cortex organotypic slice cultures and whether neural progenitor cells could repair the injured cortex. This was interesting for me as I will be able to modify the injury paradigm and use it for experiments to examine white matter damage. The third poster was by J de Vellis (UCLA, Los Angeles). In their poster they demonstrated that following an excitooxic injury of the pervientriuclar white matter, migration from the SVZ of oligodendrocyte precursors was reduced whilst following treatment with a combination of IGF1 and transferring this was alleviated and the number of Ki67+ cells significantly increased as result which could be due to neuroprotection and repopulation of the oligodendrocyte precursors via increased migration out from the SVZ

The final day of the conference was for me one of the busiest with a number of interesting and important poster sessions revolving around the theme of perinatal brain injury and repair. Once again there were too many posters to comment upon, however there are a couple worth mentioning. C Zhu's group (University Gothenburg, Sweden) had two posters side by side. The first was illustrating the beneficial neuroprotective effect of lithium following neonatal hypoxia/ischemia with a reduction in tissue loss and reduced caspase activation. Using the same lesion model the group also used a novel application of nitric oxide inhalation to significantly reduce tissue loss in the brain. Interestingly this result was more pronounced in males than females. This gender bias in the protection from injury was interesting because there was a poster from S Mayoral (Stanford University, California) who illustrated differences in sex steroids modulates the extent of damage following neonatal hypoxic brain injury.

As in previous years, the centre of the conference hall is filled with exhibitors trying to sell their wares. There were a number of interesting stands, particularly the Hamilton syringe company who have a new adaptor kit to attach glass pulled needles to the end of the their syringes. Leica were displaying a new cryostat device that allows tissue sections of 2μm to be cut, and a new piece of stereotaxic apparatus that uses the Paxinos rat brain atlas to allow very precise injections to be made. Of particular interest to me was the Nikon stand which was displaying the top 100 winners from this years Nikon Small World competition in which I managed to get an Image of Distinction.

Society for Neuroscience meetings remain the largest scientific meetings that neuroscientist and clinicians attend during their careers with some attending each year without fail whilst others only attending every few years. I had not been for over 4 years and it was a pleasure to return to this meeting. For every symposia, lecture and poster that I attended there were at least a dozen more that would have captivated my interest. This years meeting was also combined with a fantastic host city, with the nightlife and architectural sights of Chicago and particularly favourable weather making SFN 2009 a particularly enjoyable scientific meeting.

Dan Webber
University of Cambridge

Exploring the Society for Neuroscience 2009 meeting, the first poster that caught my attention was on the measurement of spontaneous-pain behaviours in rodent animal models.

This work, by Jeffrey Mogil's group (McGill University, Montreal), is particularly relevant to neuropathic pain states in humans, as spontaneous pain (continuous or paroxysmal) is the most prevalent symptom in those patients. Traditionally, either due to technical difficulties or lack of well-characterised tests, assessment of pain behaviours in animals is mostly based on measurement of responses

to evoked stimuli. While this correlates with hypersensitivity of the sensory nervous system and is of certain value for the pharmacological evaluation of treatments, it does not fully reflect the human condition. This group conducted a thorough study of the use of some of the most commonly used spontaneous pain outcomes and presented evidence that these outcomes have a different time-course when compared with evoked-pain tests, show significant variability between different strains. Furthermore, they found that in contrast to evoked-pain tests, spontaneous pain assays do not always reflect restoration of normal function when using well-established neuropathic pain treatments. They conclude that the subject of spontaneous pain measurement in animals should be approached carefully and emphasize the need for new assessment methods.

Another poster more directly related to my own research was presented by Nikita Gamper's group (University of Leeds, Leeds). This work examined the contribution of KCNQ voltage-gated potassium channel subunits (more known for their putative role in epilepsy) in the establishment of neuropathic pain. The authors provided very interesting expression data for those channels in sensory neurons, either in the naïve system or following peripheral injury. They found significant down-regulation of those proteins in pain states, which they linked to functional consequences on hyperexcitability of sensory neurons. Finally, they identified upstream targets that transcriptionally regulate KCNQ channels and examined the regulation of those after injury as well. Targeting those targets could provide new insight on the understanding/control of pain.

Between posters I attended the presidential lecture given by the Nobel prize winner Eric Kandel titled 'on the perpetuation of long-term memory'. Having been a little bit late, I was lucky enough to find an empty seat in the packed amphitheatre. This was an enlightening talk which packed a whole lifetime worth of experiments, from the discovery of the molecular constituents of memory-dependant behaviours in worms and flies, to the formation of sophisticated synaptic pathways in the mammalian brain. It is quite challenging to describe such complex interactions to the non-specialised viewer, but Kandel managed to make it seem simple – though it certainly was not. This lecture was definitely one of the highlights of the conference, one that almost definitely inspired younger scientists.

On the last day I presented my own research in which we thoroughly characterised the expression of a relatively unknown potassium channel, Kv9.1, and interacting partners Kv2.1 and Kv2.2 in rat sensory neurons. Additionally, we demonstrated robust and long-lasting transcriptional down-regulation of these subunits in models of traumatic neuropathy and proposed that this loss of function may be linked to emergence of spontaneous neuronal activity and development of neuropathic pain. During my presentation, I was able to meet a number of well-respected researchers and discuss my work and future goals with them, opening the way for a number of collaborations. It was pleasant to see familiar faces that I remembered from last year's meeting – you certainly start to feel part of a bigger, global, group. Additionally, I encountered individuals that work on related targets in different contexts, with whom I shared information and insight. Overall, my SfN presentation was a very helpful and worthwhile experience that gave me more confidence in my research and also benefited me from a more practical point of view. I shall be definitely repeating it!

Christoforos Tsantoulas
The Wolfson Centre for Age-Related Diseases
Kings College London

16th International Conference on Neural Information Processing

1–5 December 2009. Bangkok, Thailand

The 16th annual International Conference on Neural Information Processing (ICONIP) brought together an interdisciplinary group, consisting of mathematical, physical and biological scientists to discuss the past, present, and future challenges and trends in the field of neural information processing.

ICONIP accepted around 400 submitted papers that have been peer review by at least 2 independent referees. Accepted papers have been published in "Springer lecture Notes in Computer Science" under the rubric Advances in Neural Information Processing. Topics of accepted submission ranged from methods and techniques for artificial neural networks, neurocomputers, brain modeling and bioinformatics applied to a large variety of problems in neuroscience, biology, medicine and engineering.

We developed a method to study neural adaptation based on nonlinear dynamical models estimated purely from experimental data. We showed in addition results of applying the method on light adapting photoreceptors in drosophila. The method is different from methods currently applied for studying neural adaptation. It offers the quantitative study of changes in the system dynamics in the time and frequency domain, which has been difficult to comprehend from other methods. By attending ICONIP, I had the chance to present the method and our results to experts in the field of neural information processing. Our paper was rated within the best 15% of all student

papers and as such, it accepted for an oral presentation. In this way, I have been able to present our work to many people at once, which initiated several further discussions directly after the presentation and in the later course of the conference.

To meet and discuss our work with people that deal with similar challenges, helped to compare other method with ours, and emphasize possibilities to improve our technique. In this way I made several contacts with people, I already know from their publications and others I just became aware of their work.

Apart from meeting people, the attendance of the conference helped me to obtained a good overview on state of the art methods and their applicability to our work.

The study of the program in advance to the conference was very beneficial. Considering the enormous amount of contributions, it would have been nearly impossible to filter out the relevant ones on the spot. I would suggest anyone to pre-select the contributions of interest in advance to the conference, especially for conferences with parallel sessions. The next time, I even plan to look up the web-sites of individuals and make notes on their general research interest before approaching them. Although, I had the chance to get in contact with many participants, being rather new in the field, this would have helped even more to target important people, not only by their current contributions. The meeting has been split in several parallel sessions and some of the most interesting sessions were overlapping. Because a fellow student of my lab attended the conference together with me, we decided to split up and attended important parallel sessions individually to discuss the presentations in the breaks or evenings. This at least allowed us to get an idea what had been part of the sessions, not attended.

In an oral presentation I presented our paper "Data Modelling of Analysis of Adaptive Changes in Fly Photoreceptors" [1]. In this work, we studied sensory adaptation in *Drosophila* photoreceptors. Adaptation is known to efficiently enable encoding of sensory information in single neurons and neural chains by tuning the neuron's input-output relationship. This it does to represent sensory information best within its limited dynamic range (0-60mV) of the neurons output. Because the underlying physiology of adaptation processes is complex and difficult to integrate into a biophysical model, we employed nonlinear system analysis as a tool for studying the neurons' underlying coding strategies.

Based on the NARMAX methodology we quantified luminance adaptation in nonlinear dynamical models that are estimated from experimental input-output measurements of fruit fly *Drosophila* R1-R6 photoreceptors. The input contained selected light patterns of naturalistic structure which has been presented at light levels ranging in logarithmic steps over 10,000 fold.

Individually estimated models from each light level were able to accurately predict the neurons voltage response within corresponding light levels. By transforming these models into the frequency domain as Generalized Frequency Response Functions, we were able to find a global model structure and a single parameter set to approximate adaptive changes by a pure change in the input gain.

Delegates were equally interested about the methodology and the results. For the reason that there are already quite a number of models of fly photoreceptors available and used by other groups, several delegates were suspicious, which advantages and new insight our approach provides. Questions asked during the session turned out to be beneficial in addressing the difficulties of our approach.

Attending ICONIP 2009 allowed me to present our ongoing work on the study of *Drosophila* photoreceptors. Despite drawing in an oral presentation the attention of other conference participants towards our project, the publication of our submitted paper will be a remaining contribution that can be viewed online.

The success of our contribution at the conference and the positive feedback towards our work reinsured our projects current direction. Critical feedback on the other hand gave me new ideas how to improve current difficulties within our methodologies.

In the course of the conference I was lucky to make some nice contacts with other researchers in the field that I hope to see again at a similar occasion.

I happily can conclude that the conference has been very exciting and was beneficial towards my work. I had a really good time and I am grateful for all support that allowed me to have this opportunity.

I would like to thank the Society of Cell Biology (BSCB) for their generous support towards my expenses for attending ICONIP 2009. The BSCB Honor Fell Travel Award has been essential for covering my travel cost and offering me this great experience. Thank you!

Uwe Friederich
University of Sheffield

1 Friederich, U., Coca, D., Billings, S. & Juusola, M. (2009), Data modelling for analysis of adaptive changes in fly photoreceptors. Lect. Notes Comp. Sci. (ICONIP 2009, Part I) 5863: 34–48

ICONIP'09 aims to bring together scientists, practitioners, and students worldwide, especially from the Asia-Pacific region, to discuss the past, present, and future challenges and trends in the field of neural information processing.

This year, ICONIP attracted approximately 400 submissions, with approximately 300 presentations accepted. These covered a large scope of topics in the areas of: methods and techniques of artificial neural networks, neurocomputers, brain modeling, neuroscience, bioinformatics, pattern recognition, intelligent information systems, quantum computation, and their numerous applications in almost all areas of science, engineering, medicine, environment, business.

My interdisciplinary research project is to study how a network of photoreceptors and inter-neurons co-process visual information through feed-forward and feedback mechanisms. We began studying this question in a simple eye of *Drosophila* by constructing a mathematical model, which describes photoreceptor voltage responses to light stimuli. This model is a biophysical model, which can show the dynamics of important molecules in the photo-

transduction cascade. The model can be used to understand how the early vision system adapts to environmental light changes and what the key molecular mechanisms are. The knowledge gained from monitoring the molecular dynamics during responses may benefit to pharmaceutical problems.

Because of the specific aim of the conference to enhance interdisciplinary collaboration, there are many parallel sessions for different topics during the conference. Over the 4 days of the conference, my main focus was to attend sessions of primary interests, namely computational neuroscience and the related applications. Though the studied subject may be on other organisms or other neurons, for example, it may be on mammalian organisms or on neurons in the cortex or deep brain, the scientific questions being studied and the potential applications are of great interests to me. Furthermore, the general methodology is also interchangeable and fallen into my interests, for example, mathematical modeling and analysis and learning algorithms. The techniques will be beneficial for developing or analysing my model further. The model may need to be parameterised to make predictions about unknown mechanisms, therefore, learning algorithms are in particular useful. This sub-session arrangement will allow me to attend sessions that will be of utmost importance to my field of work, but also ensures that I am kept up-to-date with the latest trends and technologies occurring in other field of computational neuroscience.

I presented my paper entitled, "Biophysical modeling of *Drosophila* photoreceptor" on the second afternoon of the conference. I was the last one in the session, right after my colleague presenting his research, which is directly related to mine. His topic was also modeling *Drosophila* photoreceptor, but using a black box method and looking the system at a more abstract way than mine. Since, our projects are studying the same system at macro and micro level respectively, in this arrangement of the talks, we were able to refer to and reinforcement each other's research topics. At the beginning of my presentation, there were some people coming specifically for my talk, indicating that lots of people in computational neuroscience are interested in biophysical mechanisms in single neuron computation.

At the end of the presentation, there were several questions fielded. These questions range from biological background of the organism to the potential use of the model, including both in medical area or engineering area. In responses to the probable applications of the model, I kept it open for discussions, because the model now is not in a complete stage and we could not know specifically what answers it could give out for our scientific questions asked. However, people showed great interests on the questions we would like to study and we got some useful advices on how to extend the model for scientific aims.

In general, there are two ways for a model to go: it could become bigger and bigger by adding more and more elements into it. One group from RIKEN Brain Science Institute led by Shiro Usui is building up a collaboration platform for this purpose of integrating existing models to large scale level. They call this project PLATO, and the key merit of this project is that people could construct

computational models using several programming languages and connect them at the I/O level with a common data format. As an example, a whole visual system model, which includes eye movement, eye optics, retinal network and visual cortex, was constructed. Their preliminary results demonstrate that the integrated model successfully simulate the signal processing flow at different stages of the visual system. However, unfortunately, until now, the model has not been used to study one specific question. The other way for a model to go is becoming more and more into detail, for example, models at molecular level. One group from University of Warwick built a model for Cerebellar Purkinje Neuron based on Hodgkin-Huxley formulism. The model also takes into consideration of the geometry properties of the neuron. Using this model, they found that Na^+/K^+ bump is taking a role in controlling intrinsic Purkinje cell firing pattern. This piece of work shares the same methodology with my research, hence I discussed a lot with the author of this paper and it turns out that we know lots of people in the field in common.

In summary, I would like to take this opportunity to thank BSCB for the award of Honor Dell Travel Award. By exposure to such an international affair, I was beaming with ground breaking research and interaction with leaders in the field of computational neuroscience from all over the world. More importantly, my research work has been published in an international standing publication, and was also critically reviewed by a good panel of international Scientists. This will provide the motivation for taking this research into the next phase and increase my confidence that the outcome of the research is heading in a positive direction.

Zhuoyi Song
University of Sheffield

BSCB Summer studentships

2009 saw the second cohort of undergraduate students enter labs to undertake summer studentships of around 8 weeks funded by the BSCB. These studentships provide valuable experience, and the scheme will continue in the coming years. Details of the 2010 round can be found on the BSCB website including full details of the application procedure. The deadline for applications is 30 April 2010.

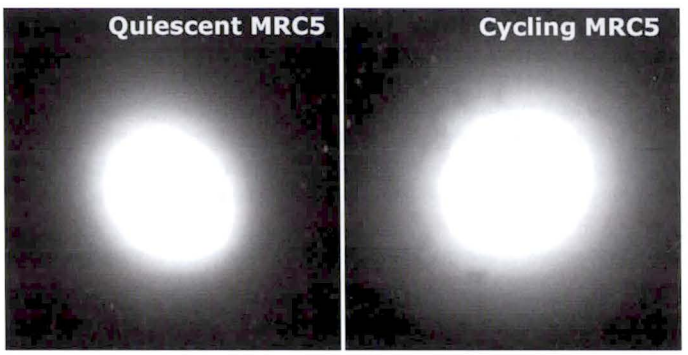
Analysis of the Interaction between Ciz1 and the Nuclear Matrix

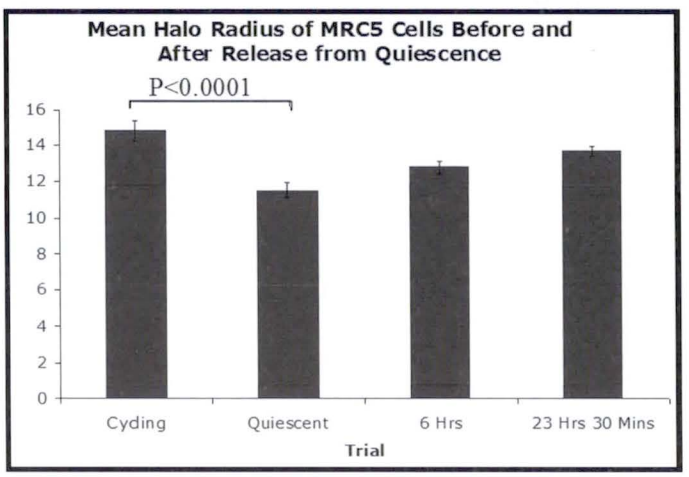
This summer I spent eight weeks in the Coverley Lab at the University of York, funded by the BSCB. I was taught a range of lab skills including mammalian cell culture, transfection and fluorescent microscopy but my main task was to set up and optimise the maximum fluorescent halo radius (MFHR) method. The MFHR method when applied to cells causes the expansion of the chromatin outside of the nucleus to form a halo like structure that reflects the chromatin loop size [1]. This is achieved by dissolving the membrane with a detergent called NP40, extracting the chromatin bound protein (including histones) with increasing salt concentrations and by nicking the DNA with UV light [2].

Initially, the MFHR method was used to determine the loop size of cycling cells for four cell lines. The halo radius of each cell line was consistent with one another - approximately 14.7μm. This converts to a loop size of 67.62 kbp [1], which is consistent with the published literature. I also investigated whether loop size changes when cells enter and exit quiescence. A reproducible and significant change in the halo radius was recorded for all cell lines tested. Towards the end of my placement, I took part in an RNAi experiment to ask whether Ciz1 plays a role in loop remodelling during the exit from quiescence. Again consistent data was attained that showed a significant change in the halo radius. Further work will be conducted in both these areas to determine the nature and source of the changes in loop size under these treatments. I have found this placement an invaluable experience and have gained much insight and enjoyment from working as part of a laboratory team and I hope to continue in this field. I would like to give special thanks to Dawn Coverely, Nikki A. Copeland and Faisal Abdel Rahman both for sharing their reagents and for helping me in the lab.

Laura M Knight
Undergraduate, Molecular Cell Biology, University of York

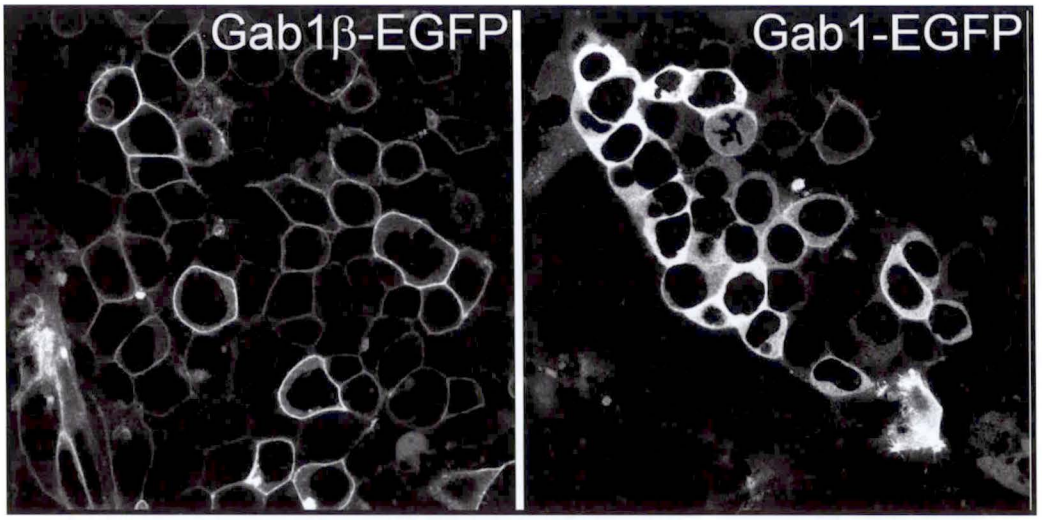
1. Buongiorno-Nardelli, M., et al., A relationship between replicon size and supercoiled loop domains in the eukaryotic genome. Nature, 1982. 298(5869): p. 100-2.
2. Lemaitre, J.M., et al., Mitotic remodeling of the replicon and chromosome structure. Cell, 2005. 123(5): p. 787-801.





Top: Quiescent and cycling MRC5 cells. Below: MFHR measurements for cycling MRC5 cells, cells 4 days in quiescence and following release from quiescence for the indicated times. A significant drop in MFHR was recorded which recovered upon re-entry to the cell cycle. Images show typical halos, which were measured vertically from the lower edge of the nucleus to the lower edge of the halo (n=between 20 and 70 nuclei per condition).

Cellular localisation of a novel signal transducer in embryonic stem cells



Above: Images obtained using confocal microscopy showing membrane and cytoplasmic localisation of Gab1 and Gab1 EGFP fusion proteins respectively, in ES cells.

Grb2-associated binder protein-1 (Gab1) is a common, adaptor-type signalling molecule that can be activated by a wide range of growth factor and cytokine receptors. Loss of Gab1 activity in vivo, in knock-out mice, is reported to lead to defects in the development and function of placenta, heart, muscle, liver and skin. This complex phenotype is presumably largely due to deficits in cellular responses that have been associated with Gab1 in vitro, such as cell proliferation, cell survival, response to stress and morphogenesis. At the molecular level, Gab1 operates as a platform for the assembly of signalling complexes at activated cell surface receptors. The C-terminal domain contains multiple protein–protein interaction motifs including binding sites for its co-adaptor protein Grb2, and the key downstream effectors, the protein tyrosine phosphatase SHP-2, and the lipid kinase phosphatidylinositol-3-kinase (PI3K). Recruitment and activation of PI3K at the plasma membrane, results in the production of phosphoinositide PIP3, which in turn, serves as a ligand for the N-terminal pleckstrin homology (PH) domain of Gab1. This positive feedback loop is thought to stabilise association of the adaptor at the plasma membrane in stimulated cells and leads to sustained activation of downstream signals – via SHP-2 and other Gab1 effectors.

In Dr Tom Burdon's lab, the group have identified a variant Gab1 protein (Gab1β) that is highly expressed in mouse embryonic stem (ES) cells and lacks the majority of the N-terminal PH domain. Remarkably, however, preliminary experiments indicated that the protein is still targeted to the plasma membrane. The aim of my project was to determine what controls Gab1β localisation in ES cells.

My first objective was to accurately define the intracellular location of endogenous Gab1 protein in ES cell lines using confocal microscopy. By comparing the distribution of Gab1 protein in wild type (WT) and Gab1β knock-out cells using a pan-Gab1 antibody, it was clear that the ES cell specific β-form is constitutively located at the periphery of cells. Moreover, a fluorescent fusion protein comprising of Gab1β tagged with EGFP was also localised at the cell membrane (see figure). In contrast, the full-length Gab1-EGFP fusion protein containing the intact PH domain was located in the cytoplasm as well as at the plasma membrane – consistent with previous reports from other laboratories.

These experiments indicate that Gab1 β contains a cellular "address" that directs it to the plasma membrane. My second objective was to identify this element in the Gab1β protein. Mutant Gab1β-EGFP fusion proteins lacking binding sites for either Grb2 or SHP-2 displayed the same cellular distribution as the WT protein, demonstrating that interactions with these Gab1 partner proteins do not influence Gab1β localisation. In contrast, fusion proteins with deletions of the N-terminus of Gab1β were cytoplasmic, indicating that some component of the membrane targeting sequence is contained at this end of the protein. Finally, stable transfection of an E-cadherin knock-out ES cell line with either E or N-cadherin cDNA expression vectors did not induce Gab1β protein levels, implying that the high levels of this adaptor in ES cells are not cadherin dependant.

The summer studentship gave me an opportunity to develop many essential scientific skills. I learned various techniques including, ES cell culture, transfection, and immunohistochemistry. Training in the use of the confocal microscope was a particularly valuable experience. Apart from that, I had a chance to analyse real scientific data and to face many of the problems or difficulties that arise in real experiments. I would recommend any student to participate in a summer project, as it will give them a realistic insight into the scientific world. It also will help in making decisions about the direction of their future scientific career.

Student: Magdalena Stasiulewicz
Supervisor: Dr Tom Burdon

Effects of formins during angiogenesis

Angiogenesis is the process of formation of new blood vessels from pre-existing ones. Uncontrolled angiogenesis makes a negative contribution during some clinical conditions such as cancer and diabetic retinopathy[3]. The process requires dramatic changes to cell shape as the endothelial cells form a new vessel[2]. The mechanisms underlying these shape changes are largely unknown.

The formin family of actin regulatory proteins have been shown to control cell elongation in a number of situations[1]. In the present research studentship, I examined the possible role of formins in angiogenesis. The aims of the 2 month studentship were 3-fold. The first was establish conditions for silencing expression of formins in endothelial cells using siRNA and to quantify this by qPCR. The second was to silence expression of isoforms of the formin family and to observe the quantitative effect of this on angiogenesis. The third was to find which of the Rho GTPases acts as the upstream signalling molecule for the formin protein being studied.

To observe the effects of silencing formin expression on angiogenesis, an *in vitro* assay was performed using Human Umbilical Vein Endothelial Cells (HUVECs) which were cultured in endothelial growth medium (EGM). HUVECs were treated with 4 independent small interfering RNAs (siRNAs) targeting the formin protein. There were two control HUVEC cultures, one treated with siRNA targeting lamin (which is known to have no effect on angiogenesis) and the other left untreated with any type of siRNA. After siRNA treatment, the cells were used in a co-culture assay of angiogenesis. Six days later, the co-culture was fixed in –20°C ethanol and immunostained using anti-human PECAM as primary antibody and anti-mouse alkaline phosphatase conjugate as secondary antibody. The vessel formation in the six samples was quantified for density of the vessels formed. After initial success, 2 other independent experiments were performed and it was observed that 2 of the 4 siRNA tranfections targeting the first formin protein resulted in over 50% reduction in vessel density as compared to the 2 control samples.

To confirm the knock-down of the first formin protein, a Q-PCR was performed. For this, the HUVECs were transfected with the 4 independent siRNAs targeting the first formin protein and there were 2 control cell cultures, just as stated above. The cells were cultured in EGM and RNA was extracted after 2 days using the Trizol extraction protocol. Reverse transcription polymerase chain reaction (RT-PCR) was performed, followed by Q-PCR using RP-II as a control for the six samples and SYBR-green as the DNA stain. The Q-PCR was performed twice with partial success on the first attempt and complete failure on the second attempt (because of manual errors). The partial results of the first attempt were quantified using the comparative Ct method and could only be used to compare the 2 control samples with effects of just one of the independent siRNAs targeting the first formin protein. These results were inconclusive because they indicated that transfection with lamin siRNA increased expression of the first formin protein by 3-fold, as compared to GeneFECTOR control. Therefore, it was decided to perform a western blot to observe the knockdown caused by the 4 independent siRNAs targeting the first formin protein. The western blot showed clear knockdown of the first formin protein being caused by the siRNAs targeting it.

To find out the upstream signalling Rho GTPase of the first formin protein, yeast two-hybrid assays were performed. For this, the GTPase binding domain (GBD) of the first formin protein was amplified using PCR and inserted into pGADT7. 16 Rho GTPases were obtained from another laboratory in pYTH6 and pYTH9 plasmids. Out of the 16 Rho GTPases, only 6 could be appropriately screened for interaction with GBD of the first formin protein. After co-transforming the yeast (AH109) with pGADT7 and pYTH6, the transformants were screened on double dropout (DDO) plates (deficient in tryptophan and leucine). The transformation efficiency was low and some of the experiments resulted in no transformants at all. The transformants were plated on triple dropout plates (deficient in histidine to observe GBD-Rho GTPase interaction) with increasing concentrations of 3-AT (which inhibits histidine biosynthesis) to increase the stringency. Out of the 6 Rho GTPases screened, one of the Rho-GTPase showed clear interaction with GBD of the first formin protein.

In all, although I faced many failures, I had a great time during the 2 month studentship and would most certainly pursue a career in research and do a PhD. I would like to thank BSCB for providing students like me with such valuable experiences. Also, I'm more than thankful to Dr. Harry Mellor for letting me obtain this experience in his lab and for being a great supervisor. I'm also very thankful to Clare Hetheridge (PhD student), Dr. Alice Scott (Post doc) and Lifie Fan (PhD student) for all their valuable time, expertise and patience to teach me some very important techniques and for being such wonderful teachers.

Maxim Saini

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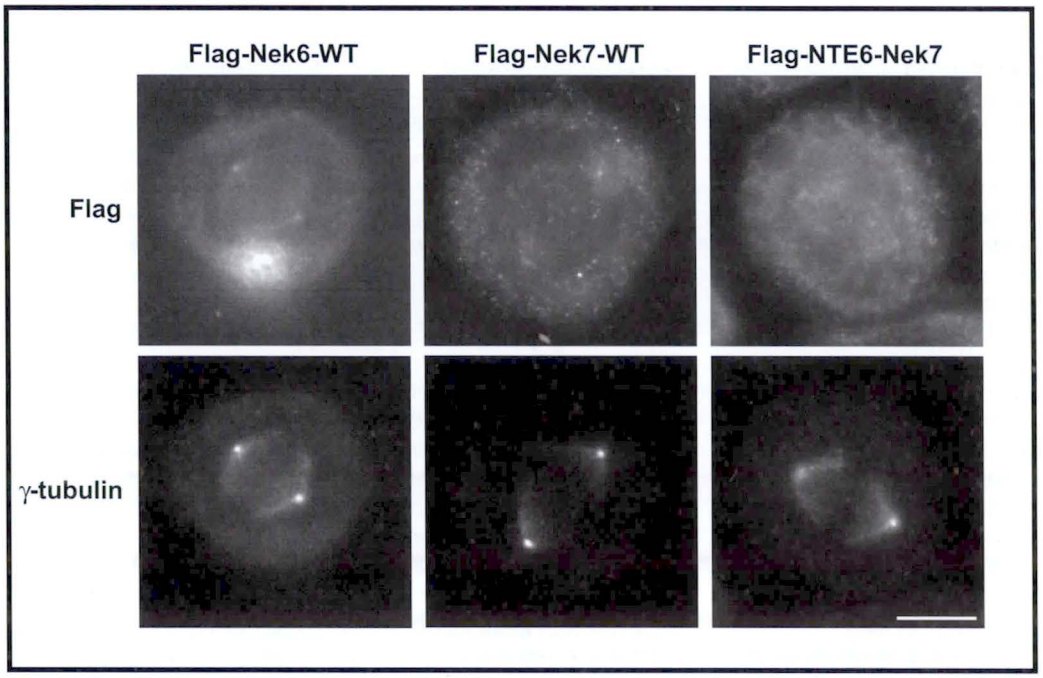
Investigating the roles of the N-terminal extensions of the cell cycle regulated Nek6 and Nek7 kinases using a chimaeric approach



Immunofluorescence microscopy was used to study the localization of wild-type and chimaeric Nek6 and Nek7 protein kinases in mitotic cells. HeLa cells transfected with the Flag-tagged proteins indicated were stained for the Flag-tag and the spindle pole marker, g-tubulin. Flag-Nek6-WT is detected on spindle microtubules whereas Flag-Nek7-WT only clealy localizes to spindle poles. Images of the Flag-NTE6-Nek7 chimaera suggesting that the N-terminal extension (NTE) of Nek6 targets the protein to spindle fibres. Scale bar, 10 mm.

Through my University studies, I have become intrigued to understand what happens when cancer develops in the human body. I was also keen to find out what biological research was really like and whether it would be a career that I'd like to follow. I was therefore excited by the possibility of getting some real experience in basic cancer cell biology through a summer studentship placement in the laboratory of Professor Andrew Fry at the University of Leicester.

Professor Fry's lab works on a family of human protein kinases, the NIMA-related kinases (or Neks), that contribute to control of cell division. Four members of this family, Nek2, Nek6, Nek7 and Nek9, play roles in mitosis (O'Regan et al, 2007). Nek6 and Nek7 are unusual in being composed of a highly-related kinase domain with just a short (30–40 residue) divergent N-terminal extension (NTE). Interestingly, although both are required to pass the metaphase-anaphase transition, they differ in their subcellular localization during mitosis (O'Regan & Fry, 2009). Moreover, putative substrates of Nek6 have been identified that are not phosphorylated by Nek7. With this in mind, the aim of my project was to test the hypothesis that it is the NTEs that determine where the proteins localise and their substrate specificity. For this, protein chimaeras in which the short NTEs were swopped had been generated creating the NTE6-Nek7 and NTE7-Nek6 fusion proteins whose behaviour in cells I would compare to the wild-type proteins.

The experimental procedures required, firstly, culture of HeLa cells and transfection with constructs expressing the different proteins. Transfection was then tested using Western blotting before either protein localizations were studied by immunofluorescence microscopy or bound proteins analysed by immunoprecipitation and Western blot. The results that I obtained confirmed that in mitotic cells wild-type Nek7 localises only to spindle poles whereas wild-type Nek6 localises to both spindle poles and spindle fibres. The NTE6-Nek7 chimaera appeared to localise mainly to the spindle fibres in much the same way as wild-type Nek6, strongly supporting the hypothesis that it is indeed the NTE that determines protein localisation. I was also able to immunoprecipitate the different proteins although, unfortunately, didn't have time to examine the coprecipitating partners.

Before I began the studentship I was unsure how I would find working in a laboratory full time as, during undergraduate practical classes, laboratory time is limited and instructions provided so leaving no need to plan your own experiments. However, my worries were quickly forgotten as straight away there was so much to learn and everyday there was something new to do and a new obstacle to overcome. The best thing about working in a laboratory is the feeling you get when something that hasn't been working starts to work, or when you find something that leads to more questions being asked that will further the understanding of the project. I have learnt many things from this experience such as having to think and decide what experiment to carry out next to proceed with the investigation. I thoroughly enjoyed my eight weeks in Professor Fry's lab and it was a shame that it came to an end so quickly. However, I really feel that I have learnt vast amounts during my time there and I now know for sure that I would love to continue in research after my Biochemistry degree. Finally, I would like to thank the BSCB for giving me this priceless opportunity and Professor Fry and Dr. Laura O'Regan for spending their valuable time and helping me throughout my project.

Sarah Sabir

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The association of BRCA1 with CtIP

Individuals with a genetic predisposition to breast cancer go on to develop the disease which is associated with a loss of either BRCA1 or 2 function. Cancer cells which lack either BRCA1 or 2 have a significantly reduced ability to carry out homologous recombination, an important mechanism for DNA repair. Because many cancer treatments involve inflicting DNA damage, it is important to understand how cells respond to DNA damage and exploit differences between normal and cancer cells to enable the selective killing of the latter.

Recent progress has identified the CtIP protein as an additional critical player in the process of homologous recombination. Importantly work from a variety of laboratories have demonstrated the importance of a CtIP-BRCA1 complex, without which HR does not occur. The association of BRCA1 with CtIP is dependent on phosphorylation of the latter on a single serine residue, serine 327.

The aim of my project was to establish in the lab, an in vitro assay to detect the association between BRCA1 and CtIP. To do this, I transfected human embryonic kidney cells (HEK293) with a cDNA encoding a tagged version of full-length CtIP, and confirmed that the cells expressed CtIP by carrying out a Western blot using an antibody directed against the FLAG epitope. Next, I transformed a bacterial expression strain with a plasmid encoding a GST fusion containing BRCA1 C-terminal BRCT domains, which contain the region of BRCA1 required for interaction with CtIP.

In order to detect the BRCA1/CtIP interaction, the HEK293 cell lysates containing over-expressed CtIP were incubated with glutathione-Sepharose beads loaded with bound GST-BRCA1-BRCT, or control beads bound with irrelevant protein. After washing the beads, specific interaction between the BRCA1-BRCT domain and CtIP was detected by SDS-PAGE electrophoresis of protein complexes specifically retained by the beads and immunoblotting with the anti-FLAG antibody. This experiment demonstrated that overexpressed CtIP associated with recombinant GST-BRCA1-BRCT, suggesting that at least some of the overexpressed CtIP is phosphorylated at serine-327 in HEK293 cells. I then used a site-directed mutagenesis (SDM) approach to mutate CtIP serine-327 to alanine, in order to create a form of CtIP which is predicted to be incapable of binding the BRCA1-BRCT domain. Unfortunately, sequencing of multiple DNA samples following SDM failed to detect any mutants, and time contraints prevented me from undertaking another round of mutagenesis and screening.

Once the negative control construct has been created and verified, this assay will be used in the future to examine the effects of putative modulators of the BRCA1-CtIP interaction, as well as for the development of prognostic indicators of breast cancer treatment effects on the HR process. Both approaches should ultimately help breast cancer patients.

I would like to thank Prof Carl Smythe for allowing me to work in his lab, Dr Richard Beniston for direct supervision, and the BSCB for the funding of my placement. This summer vacation studentship has provided me with first-hand experience of real cutting-edge research work and I am very grateful to have had this opportunity.

Matthew Robson

Professor Carl Smythe's lab, University of Sheffield.

Keeping Libel Laws Out of Science – Fighting for our Right to Debate

Jay Stone

If you asked my friends why they thought I was writing for the BCSB they would undoubtedly say it is because I have an overactive mind and needed a medium in which to express my thoughts and ideas, they would also probably express their gratitude to the BSCB as I am now not harassing them with my ponderings.

Whilst this is true in some respects (people in my lab can now drink their coffee and eat digestives in peace), the main reason I volunteered to write for the BSCB was because I wanted to get experience in scientific communication which I believe to be vital if you want a successful career.

During my endeavors to explore science communication, I came across a charity called 'Sense about Science' (SAS), a non-profit charity trust that work with over 2,000 scientists and civic groups to respond to misrepresentations of science in the public domain. SAS believe in good science communication promoting public understanding to prevent panic and confusion. The topical publications they produce such as 'Making sense of GM' are easy to read and appeal to all levels of background knowledge. They are available on their website and are often requested by schools, hospitals and media companies for learning or research resources. During a recent internship with SAS I was lucky enough to get involved with their latest hot potato topic – libel laws in science.

In science healthy debate and discussion of each other's work is crucial. We all read papers and discuss what we think about them, whether we agree with their controls, their statistical analysis, whether we would have drawn the same conclusions. It is what drives our work forward.

We put so much emphasis on having a fresh pair of eyes on our work that we have even designed the peer review system around it. Science is meant to be open for all to see, warts and all!

Libel laws are designed to protect us from false accusation and defamation of character. The general consensus is that if you are telling the truth and have evidence support your claims then you can't be sued for libel, you have every right to say or print the truth. So this should mean libel laws protect the good science and punish the bad, right? Well it doesn't appear to be that straight forward and last years court battle of Simon Singh vs The British Chiropractic Society proved to be the catalyst in the reaction. The opinion now is that these libel laws work to protect the rich companies against comments they don't want the public to hear. Libel laws are silencing scientific debate and worse still are preventing important information getting into the public domain.

Realising that this issue was not going to be resolved on its own and that it needed urgent

Case One: Simon Singh vs The British Chiropractic Society

Simon Singh is a science writer specialising in debunking false scientific claims and standing up to quackery.

Simon and Edzard Ernst, the worlds first professor of complementary medicine co-authored a book titled 'Trick or Treatment? Alternative Medicine on Trial'. In this book Prof. Ernst analysed over 70 studies exploring the benefits of chiropractic therapy in conditions unrelated to the back. He found there was no evidence to suggest that chiropractors could treat diseases such as colic, asthma or deafness and in fact there were some serious side effects that could result from chiropractic treatment.

Simon wrote an opinion article in the *Guardian* titled 'Beware the spinal trap' stating just this and stressing the importance of weighing up possible benefits against the possible side effects ranging from numbness and headaches to fatal vetebrea dissection and stroke. After the article was published the British Chiropractic Society sued Simon for libel. This court case is still ongoing running up what I can only imagine to be an astonishing financial bill.

Case Two: Ben Goldacre and The Guardian vs Matthias Rath

Ben Goldacre is medical doctor who writes a weekly column called 'Bad Science' for the *Guardian*. He has won many awards for his articles, which aim to expose dodgy science claims and media hype by journalists.

Back in 2007 Dr. Goldacre wrote some articles reporting on the concern he felt about Matthias Rath, a medical doctor who believes nutritional supplements can be used to treat disease. Dr. Goldacre voiced his concern over the promotion of this theory in Africa as a treatment for HIV, he claimed that Dr. Rath was promoting his vitamins as a better treatment then the conventional antiviral's and so was responsible for deaths of HIV patients.

Dr. Rath took Dr. Goldacre and the *Guardian* to the courts and attempted to sue him for libel despite Dr. Goldacre having checked all his sources and had evidence to back his statements. Thankfully Dr. Rath lost his case but the costs of libel action means that the *Guardian* are still thousands of pounds out of pocket.

You can win your case and still lose!

Case Three: Dr. Wilmshurst vs NMT Medical Inc

Peter Wilmshurst is a consultant cardiologist who is highly regarded by his peers. His libel case is one of blatant libel tourism and is one of the main issues the campaign is fighting to address.

Whilst attending a medical conference in America Dr. Wilmshurst mentioned his opinions on a new heart implant device called Starflex made by Boston based manufacturer NMT Medical Inc. These opinions were published in an online American journal called *Heartwire*.

On returning to the UK Dr. Wilmshurst found himself being summoned to court for libel action. NMT had not sued the journal *Heartwire* as they could not under US libel law but instead claimed they could sue Dr. Wilmshurst personally in the UK as the journal is online so could have been read in this country.

If Dr. Wilmshurst loses his case he faces bankruptcy but he says he will fight for the right of free speech to protect his patients.

attention SAS, English PEN and Index on Censorship joined together to start a campaign to reform the UK libel laws and I was lucky enough to attend the launch in December last year.

Standing in the conference room listening to the likes of Dara O'Briain, Nick Ross and SAS managing director Tracy Brown voice their anger and dismay about our UK libel system was inspiring and really hit home for me how much this issue can affect so many people in different professions that interact with the public.

I started to think about how long these issues might have been bubbling under the surface. How many scientists have thought twice about publishing their findings because it went against the grain of what was originally thought? How many journalists have re-worded an article or even dropped a story for the fear of being sued for libel and not being able to afford to fight their corner? I am sure for many not publishing something is better then going through with it and then having to retract it which would give the public the impression they were lying.

I also started to think about the times I had heard science being critised in the media by companies who had an interest in how the data was perceived. Take the review last year into the nutritional value of organic food vs 'normal' food. This well conducted meta-analysis by Dr Alan Dangour, a nutrionist at the London School of Hygeine and Tropical Medicine showed there was no nutritional benefit to eating organic food. Immediately there was a fierce public reaction and Dr. Dangour was accussed of dishonesty and incompetence. I remember watching some of the coverage on BBC breakfast failing to understand why people were so angry, now the mother who is earning minimum wage doesn't need to feel guilty for buying Tesco basic baked beans, isn't that a good thing? People could still buy organic if they chose too, Dr. Dangour wasn't saying they couldn't or shouldn't was he?

The point is, we as scientists have a duty to report what we find. May this be our own result or pointing out the flaws in other work it must be done to make sure everything is clear to society and nobody is duped into thinking something is a miracle cure or worrying that what they can afford isn't good enough. If libel laws are preventing us from doing this then we need to do something about it.

Over 19,000 people including performers Jonathon Ross and

Stephan Fry, Sir Mark Walport; director of the Wellcome trust, Lord Rees of Ludlow; President of the Royal Society and myself (I realise I do not sound impressive following this crowd... maybe I should have mentioned myself first...) have signed the petition but the more people who sign the bigger our voice will be so I urge all of you who haven't yet to go to the website and make your pledge. I do not want to sound like a cheesy sports coach or a tesco advert but every signature counts and together we can make a difference!

The first key to wisdom is assiduous and frequent questioning ... For by doubting we come to inquiry, and by inquiry we arrive at truth - Peter Abelard

http://www.libelreform.org/sign

Jay Stone

BSCB Calendar of Related Meetings

2010

6–9 May 2010
Cell guidance signals in cancer
Camogli - Portofino Vetta, Italy
www.embo.org

13–14 May 2010
Lysosomes in health and disease
Charles Darwin House, London,
www.biochemistry.org

18 May – 21 May
Advanced Light Microscopy Techniques and Their Applications
10th International ELMI Meeting
EMBL Heidelberg, Germany
www.embo.org

8–10 Jun 2010
Regulatory Networks in Immunity and Inflammation
Napa Valley, California
www.abcam.com/napaimmunology

8–10 June 2010
Post-transcriptional control: mRNA translation, localization and turnover
University of Edinburgh
www.biochemistry.org

14-16 June 2010
Development and stem cells in the pancreas
Stockholm, Sweden
www.embo.org

17-27 June 2010
Electron microscopy and stereology in cell biology
Oslo, Norway
www.embo.org

19-24 June 2010
EMBO/FEBS Lecture Course on the cytoskeleton in development and pathology
Djurhamn, Sweden
www.embo.org

19-23 June 2010
International meeting on chromosome segregation and aneuploidy
Royal College of Surgeons, Edinburgh
www.biochemistry.org or www.embo.org

19–24 June 2010
The cytoskeleton in development and pathology
Djurhamn, Sweden
www.embo.org

28–30 June 2010
The Physical Cell – In search of the design principles of life
University College, London
www.ucl.ac.uk/PhysCell2010/

28 June – 01 July 2010
Microscience 2010
Excel Centre, London
www.rms.org.uk

22–27 Aug 2010
14th International Congress of Immunology
Kobe, Japan
http://www.ici2010.org/

4–7 Sept 2010
The EMBO Meeting 2010
Barcelona, Spain
www.embo.org

8-10 September 2010
4th European conference on tetraspanins
University of Birmingham, UK
www.biochemistry.org

8-12 September 2010
Harden conference: Autophagy: from molecules to disease
Royal Agricultural College, UK
www.biochemistry.org

11-15 September 2010
Cell Division: time and space
Roscoff, FR
www.cnrs.fr/insb/cjm/2010/barral_e.html

22-25 September 2010
Chemical Biology 2010
Heidelberg, Germany
www.embo.org

3-8 October 2010
ESF-EMBO Symposium: Emergent properties of the cytoskeleton: molecules to cells
Sant Feliu de Guixols, Spain
www.embo.org

BSCB Autumn meeting 2010

Cell organisation through the cell cycle
5–7 September, 2010
St Catherine's College, Oxford.
Organising committee: Alison Lloyd, Buzz Baum, Gwyn Gould, Iain Hagan

See page 3 and www.bscb.org for more details

3-8 October 2010
Towards a comprehensive understanding of endoplasmic reticulum functions
Gerona, Spain
www.embo.org

9-14 October 2010
Cell biology meets microbiology
Krakow, Poland
www.embo.org

14-17 October 2010
Biochemistry and cell biology of ESCRTs in health and disease
Snowbird, Utah
http://www.asbmb.org/pagesm.aspx?id=3554

28-31 October 2010
Biochemistry of membrane traffic: secretory and endocytic pathways
Tahoe City, CA
www.asbmb.org/pagesm.aspx?id=3382

11-12 November 2010
mTOR signalling in health and disease
Charles Darwin House, London,
www.biochemistry.org

21-26 November 2010
ESF-EMBO Symposium: Molecular perspectives on protein-protein interactions
Sant Feliu de Guixols, Spain
www.embo.org

11–15 December 2010
ASCB 50th Annual Meeting
Philadelphia, PA
www.ascb.org

2011

30 March-1 April 2011
Cellular cytoskeletal motor proteins
Wellcome Trust, Hinxton, Cambridge, UK
www.biochemistry.org

3-4 August 2011
From beads on a string to the pearls of regulation: the structure and dynamics of chromatin.
A joint Biochemical Society / Wellcome Trust conference
Wellcome Trust, Hinxton, Cambridge, UK
www.biochemistry.org

31 August-2 September 2011
Dynamics within and between proteins
University of Essex
www.biochemistry.org

Biochemical Society Conferences

8–12 September 2010
Royal Agricultural College, UK

DEADLINES:
Abstract submission 8 JUNE 2010
Earlybird application 8 JUNE 2010

Autophagy: from molecules to disease

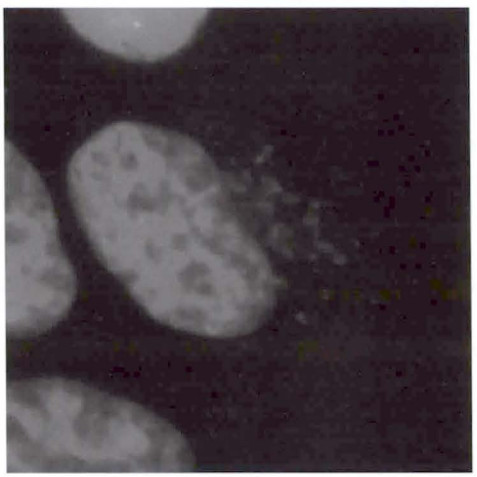

Image kindly supplied by Jon Lane (University of Bristol, UK)

Organizers:
Sharon Tooze
Jon Lane

Medals:

Grahame Hardie - Award Lecture
Peter Cullen - The Morton Lecture

Speakers:

Adi Kimchi
Aimee Edinger
Andrew Thorburn
Anne Simonsen
Beth Levine
David Rubinsztein
Deborah Goberdhan
Eileen White
Francesco Cecconi
Fulvio Reggiori
Herbert Virgin
Ivan Dikic
Kevin Ryan
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Honor Fell/Company of Biologists Travel Awards

Honor FellTravel Awards are sponsored by the Company of Biologists (the publishers of ***The Journal of Cell Science*** and ***Development)*** and they provide financial support for BSCB members at the beginning of their research careers to attend meetings. Applications are considered for any meeting relevant to cell biology. The amount of the award depends on the location of the meeting. Awards will be up to **£300** for UK meetings (except for BSCB Spring Meeting for which the full registration and accommodation costs will be made), up to **£400** for European meetings and up to **£500** for meetings in the rest of the world.

The following rules apply:

- Awards are normally made to those in the early stages of their careers (students and postdocs)
- Applicants must have been a member for at least a year (or be a PhD student in their first year of study).
- No applicant will receive more than one award per calendar year and three *in toto*
- The applicant must be contributing a poster or a talk.

No lab may receive more than £1000 per calendar year.
Awards are discretionary and subject to available funds

All applications must contain the following:

- the completed and signed application form (below)
- a copy of the abstract being presented
- a copy of the completed meeting registration form
- **proof of registration, travel and any other costs claimed**

Applications should be sent to:

Ewald Hettema
Dept. of Molecular Biology and Biotehnology
University of Sheffield
Firth Court, Western Bank, Sheffield S10 2TN

Application for Honor Fell/Company of Biologists Travel Award

Please complete, print out and send to Jordan Raff at the address above together with supporting information

Full name and work/lab address:
.

Email:

Age: BSCB Memb. No:

I have been a member for years

Years of previous Honor Fell /COBTravel Awards:

Degree(s) (dates):

Present Position:

Meeting for which application is made:
title/place/date:

Expenses claimed:

Travel:

Accommodation:

Registration:

Have you submitted any other applications for financial support? **YES/NO** (delete as applicable)
If YES, please give details including, source, amounts and whether these monies are known to be forthcoming.

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This applicant requires these funds and is worthy of support. I recognise that in the event of non-attendance at the meeting, the applicant must return the monies to the BSCB and I accept the responsibility to reimburse BSCB if the applicant does not return the funds.

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